AD-A009 593

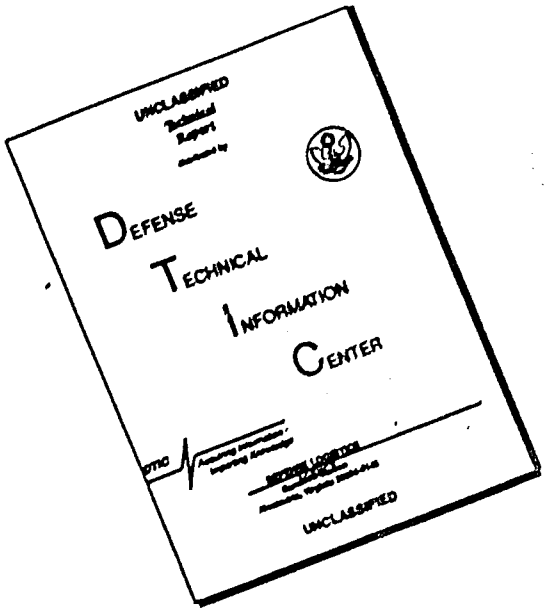
EFFECTS OF THE ABNORMAL ACCELERATORY ENVIRONMENT OF FLIGHT

Kent K. Gillingham, et al

School of Aerospace Medicine
Brooks Air Force Base, Texas

December 1974

DISCLAIMER NOTICE



THIS DOCUMENT IS BEST QUALITY AVAILABLE. THE COPY FURNISHED TO DTIC CONTAINED A SIGNIFICANT NUMBER OF PAGES WHICH DO NOT REPRODUCE LEGIBLY.

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1 REPORT NUMBER Aeromedical Review 10-74	2 GOVT ACCESSION NO.	3 RECIPIENT'S CATALOG NUMBER AD-A009 593
4. TITLE (and Subtitle) EFFECTS OF THE ABNORMAL ACCELERATORY ENVIRONMENT OF FLIGHT		5 TYPE OF REPORT & PERIOD COVERED Final - January 1974 to June 1974
		6 PERFORMING ORG. REPORT NUMBER SAM-TR-74-57
7. AUTHOR(s) Kent K. Gillingham, M.D., Ph.D. Robert W. Krutz, Jr., Major, USAF, BSC		8 CONTRACT OR GRANT NUMBER(s)
9 PERFORMING ORGANIZATION NAME AND ADDRESS USAF School of Aerospace Medicine (VNB) Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas		10 PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 9993ZSGO
11 CONTROLLING OFFICE NAME AND ADDRESS USAF School of Aerospace Medicine (VNB) Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas		12 REPORT DATE December 1974 13 NUMBER OF PAGES [illegible]
14 MONITORING AGENCY NAME & ADDRESS(if different from Controlling Office)		15 SECURITY CLASS. (of this report) Unclassified 15a DECLASSIFICATION/DOWNGRADING SCHEDULE

16 DISTRIBUTION STATEMENT (of this Report)
Approved for public release; distribution unlimited.

17 DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18 SUPPLEMENTARY NOTES

19 KEY WORDS (Continue on reverse side if necessary and identify by block number)

Acceleration	Spatial disorientation
G stress	Motion sickness
G tolerance	Vestibular function
G protection	

20 ABSTRACT (Continue on reverse side if necessary and identify by block number)

A basic description of the physical nomenclature relating to the motional environment of flight is followed by discussions of the physiologic effects of that environment on the human cardiovascular and vestibular system. The effects of $+G_z$ stress and the various means of protecting aircrew against such stress are given special emphasis. The mechanisms of spatial disorientation and motion sickness and the appropriate countermeasures are also presented.

DD FORM 1 JAN 73 1473 EDI

UNCLASSIFIED PRICES SUBJECT TO CHANGE

(SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered))

NOTICES

This final report was submitted by personnel of the Biodynamics Branch, Environmental Sciences Division, USAF School of Aerospace Medicine, Aerospace Medical Division, AFSC, Brooks Air Force Base, Texas, under job order 9993ZSGO. Dr. Gillingham was primarily responsible for writing the sections dealing with linear and angular motion, perception of motion and position, spatial disorientation, and motion sickness; Dr. Krutz authored those sections dealing with the effects of short-duration $+G_z$, high sustained $+G_z$, $-G_z$, and $\pm G_x$.

When U.S. Government drawings, specifications, or other data are used for any purpose other than a definitely related Government procurement operation, the Government thereby incurs no responsibility nor any obligation whatsoever; and the fact that the Government may have formulated, furnished, or in any way supplied the said drawings, specifications, or other data is not to be regarded by implication or otherwise, as in any manner licensing the holder or any other person or corporation, or conveying any rights or permission to manufacture, use, or sell any patented invention that may in any way be related thereto.

This report has been reviewed and cleared for open publication and/or public release by the appropriate Office of Information (OI) in accordance with AFR 190-17 and DODD 5230.9. There is no objection to unlimited distribution of this report to the public at large, or by DDC to the National Technical Information Service (NTIS).

This aeromedical review has been reviewed and is approved for publication.

Kent K. Gillingham

KENT K. GILLINGHAM, M.D., Ph.D.
Project Scientist

Robert W. Krutz Jr.

ROBERT W. KRUTZ, JR., Major, USAF, BSC
Project Scientist

Evan R. Goltra

EVAN R. GOLTRA, Colonel, USAF, MC
Commander

CONTENTS

CONTENTS (continued)

FIGURES

CONTENTS (continued)

CONTENTS (continued)

CONTENTS (continued)

EFFECTS OF THE ABNORMAL ACCELERATORY ENVIRONMENT OF FLIGHT

INTRODUCTION

A thorough understanding of the effects of acceleration on the human body is important to those involved in aviation medicine and physiology for two main reasons: (1) a pilot's performance in aerial combat, during which high, sustained acceleratory forces are experienced, is limited by the functioning of his cardiovascular and pulmonary systems; and (2) the abnormal acceleratory environment of flight contributes to the development of spatial disorientation (pilot vertigo) in instrument and formation flying. These considerations will become even more important as aircraft with greater maneuverability, such as the F-15 and the Lightweight Fighter, come into the Air Force inventory. To illustrate the increased maneuverability of the upcoming generation of aircraft, the theoretical G loads pulled by an F-15 during a simulated air-combat engagement are plotted in Figure 1. The G forces obtained in an actual engagement by an F-4E are presented for comparison.

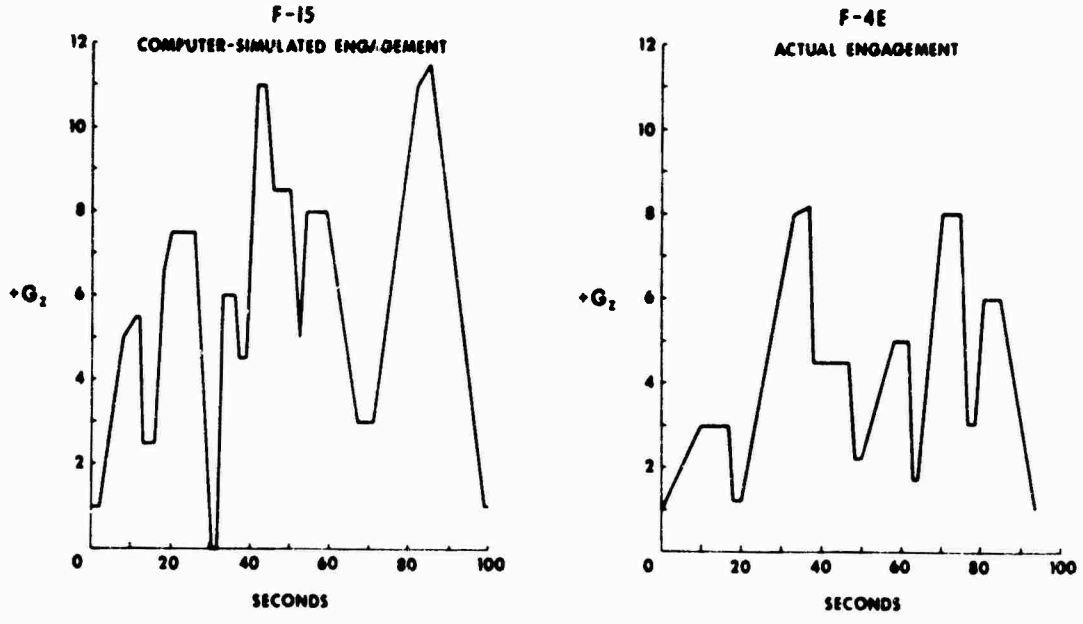


Figure 1. G forces produced in aerial combat.

LINEAR AND ANGULAR MOTION

The two kinds of motion are: linear motion or motion of translation, and angular motion or motion of rotation (Table 1). Linear motion can be further categorized as rectilinear, meaning motion in a straight line, or curvilinear, meaning motion in a curved path. Unless stated to the contrary, linear motion in this discussion will be taken to mean rectilinear motion.

TABLE 1. LINEAR AND ANGULAR MOTION--SYMBOLS AND UNITS

Motion parameter	Linear		Angular	
	Symbols	Units	Symbols	Units
Displacement	s	meters (m, feet (ft)	θ	degrees (^{o}) radians (rad) (1 rad = $360^o/2\pi \approx 57.3^o$)
Velocity	v, $\dot{s}$	m/sec ft/sec	ω, $\dot{\theta}$	^{o}/sec rad/sec
Acceleration	a, $\dot{v}$, $\ddot{s}$	m/sec^2 ft/sec^2 g (1 g = 9.81 m/sec^2 or 32.2 ft/sec^2)	α, $\dot{\omega}$, $\ddot{\theta}$	$^o/sec^2$ rad/sec^2
Jerk	j, $\dot{a}$, $\ddot{v}$, $\dddot{s}$	m/sec^3 ft/sec^3 g/sec	γ, $\dot{\alpha}$, $\ddot{\omega}$, $\dddot{\theta}$	$^o/sec^3$ rad/sec^3

Linear Motion

The basic parameter of linear motion is linear displacement, and the other parameters of motion (velocity, acceleration, and jerk) are derived from the concept of displacement. Linear displacement, s, is the distance and direction of the object under consideration from some reference point. As such, it is a vector quantity, having both magnitude and direction. The position of an aircraft located at 25 nautical miles on the 150° radial of the San Antonio vortac, for example, gives complete information about the displacement of the aircraft from the navigational facility serving as the reference point. Other commonly used units of magnitude of linear displacement are statute miles, feet, meters, and centimeters.

When linear displacement is changed during a period of time, another vector quantity, linear velocity, occurs. The formula for calculating the mean linear velocity, v, during time interval Δt is

$$v = \frac{s_2 - s_1}{\Delta t}$$

where s_1 = initial linear displacement

and s_2 = final linear displacement.

An aircraft which travels from San Antonio to New Orleans in one hour, for example, moves with a mean linear velocity of 475 knots (nautical miles per hour) in an easterly direction. Statute miles per hour, feet per second, and meters per second are other units of linear speed, the magnitude of linear velocity. Frequently it is important to describe linear velocity at a particular instant in time, i.e., as Δt approaches zero. In this situation one speaks of instantaneous linear velocity, $\dot{s}$ (pronounced "ess-dot"), which is the first derivative of displacement with respect to time, $\frac{ds}{dt}$.

When the linear velocity of a body changes over time, the difference in velocity, divided by the time required for the moving body to make the change, gives its mean linear acceleration, a.

The formula $$a = \frac{v_2 - v_1}{\Delta t}$$

where v_1 = initial velocity

v_2 = final velocity

and Δt = elapsed time

is used to calculate the mean linear acceleration, which, like displacement and velocity, is a vector quantity with magnitude and direction. Acceleration is thus the rate of change of velocity, just as velocity is the rate of change of displacement. Appropriate units for the magnitude of linear acceleration are usually feet per second squared (ft/sec^2) or meters per second squared (m/sec^2). Consider an aircraft which accelerates from a dead stop to a velocity of 200 ft/sec in 10 seconds; the mean linear acceleration is (200-0) ÷ 10, or 20 ft/sec^2. The instantaneous linear acceleration, $\ddot{s}$ ("ess-double-dot") or $\dot{v}$, is the second derivative of displacement or the first derivative of velocity, $\frac{d^2s}{dt^2}$ or $\frac{dv}{dt}$, respectively.

A very useful unit of linear acceleration is the g*, which is equal to g_o, the amount of acceleration exhibited by a freely falling body near the surface of the earth. One g is therefore equal to 32.2 ft/sec^2 or 9.81 m/sec^2. To convert values of linear acceleration given in ft/sec^2 or m/sec^2 into g-units, simply divide by 32.2 or 9.81, respectively. In the previous example, in which an aircraft underwent a mean linear acceleration of 20 ft/sec^2, one divides 20 ft/sec^2 by 32.2 ft/sec^2 per g, to obtain 0.62 g.

A special type of linear acceleration, radial or centripetal acceleration, results in curvilinear, usually circular, motion. The acceleration acts along the line represented by the radius of the curve and is directed toward the center of curvature. Its effect is a continuous redirection of the linear velocity, in this case called tangential velocity, of the body subjected to the acceleration. Examples of this type of linear acceleration occur when an aircraft pulls out of a dive after firing on a ground target, or engages an enemy in a circular path in aerial combat. The value of the centripetal acceleration, a_c, can be calculated if one knows the tangential velocity, v_t, and the radius, r, of the curved path followed:

$$a_c = \frac{v_t^2}{r}.$$

---

*The reader should be made aware of a continuing controversy that exists in the field of acceleration physiology over the use of g and/or G as units of acceleration, inertial force, or both. In the opinion of the writers, the system proposed by Hixson, Niven, and Correia, in their monograph Kinematics Nomenclature for Physiological Acceleration, provides the most logically coherent approach and allows for the greatest precision in usage; we shall adopt their system, slightly modified, for use in this review.

As an example, we can calculate the centripetal acceleration of an aircraft traveling at 1000 ft/sec (approximately mach 0.93 at sea level) and having a radius of turn of 5000 ft : $1000^2 \div 5000$ gives a value of 200 ft/sec^2 for the centripetal acceleration. This value, when divided by 32.2, comes out to 6.2 g.

For certain applications the concept of tangential acceleration, the rate of change of tangential velocity, is useful; the formula for calculating the magnitude of the mean tangential linear acceleration from the initial and final tangential velocities is analogous to the formula given above for the calculation of conventional mean linear acceleration. Occasionally one needs to find the vector sum of centripetal and tangential acceleration, as in the case of a centrifuge coming up to speed. Since the centripetal acceleration, a_c, always makes a right angle with the tangential acceleration, a_t, the net acceleration, a_n, is simply the hypotenuse of a right triangle having sides a_c and a_t:

$$a_n = \sqrt{a_c^2 + a_t^2}$$

and the angle of a_n with respect to a_t is the angle whose tangent is a_c/a_t.

One can go another step in the derivation of linear motion parameters by obtaining the rate of change of acceleration. This quantity, j, is known as linear jerk or jolt. Mean linear jerk is calculated in the following way:

$$j = \frac{a_2 - a_1}{\Delta t}$$

where a_1 = initial acceleration

a_2 = final acceleration

and Δt = elapsed time.

Instantaneous linear jerk, $\dddot{s}$ or $\dot{a}$, is the third derivative of linear displacement or the first derivative of linear acceleration with respect to time; i.e., $\frac{d^3s}{dt^3}$ or $\frac{da}{dt}$, respectively. Although units of ft/sec^3 or m/sec^3 can be used in conjunction with measurements of linear jerk, it is generally more useful to speak in terms of the g-onset rate, measured in g-units per

second (g/sec). This unit of linear jerk is particularly convenient to use when one is discussing performance characteristics of combat aircraft, escape systems, and centrifuges, as well as the qualities of impacts.

Angular Motion

The derivation of the parameters of angular motion follows in a parallel fashion the scheme used to derive the parameters of linear motion. The basic parameter of angular motion is angular displacement. For a body to be able to undergo angular displacement it must be polarized; i.e., it must have a front and a back, which is to say that it can face, or be pointed in, a particular direction. A simple example of angular displacement is seen in a person facing east, in which case his angular displacement is 90° clockwise from the reference direction, north. Notice that the angular displacement, like linear displacement, is a vector quantity, having both magnitude (90°) and direction (clockwise). Angular displacement, symbolized by θ, is generally measured in degrees, revolutions (1 revolution = 360°), or radians (1 radian = $360^{\circ}/2\pi = 57.3^{\circ}$). The radian is a particularly convenient unit to use when dealing with circular motion (e.g., motion of a centrifuge) because one needs only to multiply the angular displacement of the system, in radians, by the length of the radius to find the value of the displacement in the circular path. This is because the radian is the angle subtended by a circular arc the same length as the radius of the circle.

Angular velocity, ω, is the rate of change of angular displacement. The mean angular velocity occurring in time interval Δt is calculated thus:

$$\omega = \frac{\theta_2 - \theta_1}{\Delta t}$$

where θ_1 = initial angular displacement

and θ_2 = final angular displacement.

Instantaneous angular velocity is $\dot{\theta}$, or $\frac{d\theta}{dt}$. As an example of angular velocity we can consider the standard-rate turn of instrument flying, in which a heading change of 180° is made in one minute. Then ω = (180-0) ÷ 60, or 3°/sec. If it were useful to do so, this angular velocity could be described as 0.5 revolutions per minute (rpm), or as 0.052 radians per second (rad/sec) (3°/sec ÷ 57.3° per radian). It should

be emphasized that the fact that a body is undergoing curvilinear motion during a turn in no way affects the calculation of the angular velocity: if an aircraft were rotated half a turn on a turntable on the ground, the same value of ω as that calculated above would be obtained if the same amount of time, one minute, were required to accomplish the half turn. It is possible, in fact, for an object to undergo very complicated curvilinear motion while undergoing no simultaneous angular motion; all that is required for this to happen is that the object maintain a constant heading, i.e., face the same direction at all times.

Because centripetal acceleration results in a system when angular velocity is coupled to a radius of action, a formula for calculating the centripetal acceleration, a_c, from the angular velocity, ω, and the radius, r, is often useful:

$$a_c = \omega^2 r$$

where ω is the angular velocity in rad/sec. One can convert readily to the formula for centripetal acceleration in terms of tangential velocity, v_t, if one remembers that

$$v_t = \omega r$$

where ω is the angular velocity in rad/sec: substitute v_t/r for ω to obtain

$$a_c = \frac{v_t^2}{r}.$$

To calculate the centripetal acceleration generated by a centrifuge having a 20-ft arm and turning at 30 rpm, one uses the angular velocity formula, converting the 30 rpm to 3.14 rad/sec, squaring to obtain 9.87, and multiplying by the 20-ft radius to arrive at 197 ft/sec^2 for the value of the centripetal acceleration. Dividing this figure by 32.2 gives 6.13 g.

The rate of change of angular velocity is <u>angular acceleration</u>, α. The mean <u>angular acceleration</u> is

$$\alpha = \frac{\omega_2 - \omega_1}{\Delta t}$$

where ω_1 = initial angular velocity

ω_2 = final angular velocity

and Δt = time interval over which angular velocity changes.

$\ddot{\theta}$, $\dot{\omega}$, $\frac{d^2\theta}{dt^2}$, and $\frac{d\omega}{dt}$ can all be used to symbolize instantaneous angular acceleration, the second derivative of angular displacement or the first derivative of angular velocity with respect to time. If a figure skater is spinning at 6 revolutions per second (2160°/sec) and then takes 2 seconds to come to a complete stop, the rate of change of angular velocity is (0-2160) ÷ 2, or $-1080^{\circ}/sec^2$, which is a mean angular deceleration of $1080^{\circ}/sec^2$. The only other commonly used unit of angular acceleration is radians per second squared (rad/sec^2). One cannot express angular acceleration in g-units, which measure magnitude of linear acceleration only.

Although not commonly used in aviation medicine, there exists another derived parameter of angular displacement, angular jerk, which is the rate of change of angular acceleration. Its description is completely analogous to that for linear jerk, but angular rather than linear symbols and units are used.

Force, Inertia, and Momentum

Nearly everyone reading this will be familiar with Newton's Law of Acceleration:

$$F = ma$$

where F = force applied to an object

m = mass of the object

and a = linear acceleration.

To describe the analogous situation pertaining to angular motion, we state that

$$M = J\alpha$$

where M = torque (or moment) applied to the rotating object

J = rotational inertia (moment of inertia) of the object

and α = angular acceleration.

Force and mass are given in newtons (n) and kilograms (kg), respectively, in the mks system of units, and pounds (lb) and slugs, respectively, in the British engineering system. Torque has dimensions of force and length, because one applies a torque as a force at a certain distance from the center of rotation. In the mks system torque is measured in newton meters (n m), and in the British engineering system, pound feet (lb ft). The corresponding units for rotational inertia are n m sec^2/rad and lb ft sec^2/rad.

Since F = ma, one can calculate the centripetal force, F_c, needed to produce a centripetal acceleration, a_c, of a mass, m:

$$F_c = ma_c$$

so $$F_c = \frac{mv_t^2}{r}$$

or $$F_c = m\omega^2 r$$

where v_t = tangential velocity

and ω = angular velocity.

Consistent with the fact that for every action there is an equal and opposite reaction is the concept of inertial force, which, from a medical standpoint, is of more consequence than the force acting to produce the acceleration. The inertial force acts to keep the body from changing its velocity; i.e., it acts in the opposite direction from the acceleration. An aircraft exerting an accelerating forward thrust on its pilot causes an inertial force, equal to the product of the pilot's mass and the acceleration, to be exerted on the back of the seat by the pilot's body. Similarly, an aircraft undergoing substantial positive centripetal acceleration as a result of lift generated in a high-speed turn causes the pilot's body to exert considerable inertial force on the bottom of the seat; and, what's more important, an inertial force is exerted downward on the blood in the pilot's body as his body is accelerated upward.

At this point it is appropriate to introduce the G, which is used to measure the strength of the gravitoinertial force environment. (Do not confuse G with $\mathcal{G}$, the symbol for the universal gravitational constant, equal to 6.70×10^{-11} n m^2/kg^2.) Strictly speaking, the G is a measure of relative weight:

$$G = \frac{W}{W_o}$$

where W = weight observed in environment under consideration

and W_o = normal weight on the surface of the earth.

For example, if a 200-lb man weighs 1000 lbs while riding on a centrifuge, we say he is in a 5-G environment, or is "pulling" 5 G. In the conventional, physical definition of weight,

$$W = ma$$

and $$W_o = mg_o,$$

where m = mass

a = acceleratory field (vector sum of actual linear acceleration plus an imaginary acceleration opposite the force of gravity)

and g_o = the standard value of the acceleration of gravity (9.81 m/sec^2 or 32.2 ft/sec^2).

In that case,

$$G = \frac{W}{W_o}$$

$$= \frac{ma}{mg_o}$$

$$= \frac{a}{g_o}.$$

Thus, we see that the ratio between the ambient acceleratory field and the standard acceleration (g_o) can also be represented in terms of G. The most satisfactory convention, in the opinion of the writers, is that which employs g as a unit of acceleration (e.g., a_c = 8 g), and reserves the dimensionless ratio of weights, G, for describing the resulting gravito-inertial force environment (e.g., a force of 8 G, or an 8-G load). Another definition of G results from an alternative definition of weight, proposed for use in gravitational biology:

$$W = \frac{1}{g_o} ma$$

whereupon $$W_o = \frac{1}{g_o} mg_o$$

$$= m.$$

In this system, then,

$$G = \frac{W}{m}, \text{ the weight-to-mass ratio;}$$

but still,

$$G = \frac{a}{g_o}$$

as in the previously described conventional system.

When in the vicinity of the surface of the earth, one feels a G force equal to 1 G in magnitude directed toward the center of the earth. If one also sustains a G force resulting from linear acceleration, the magnitude and direction of the resultant gravitoinertial G force can be calculated by adding vectorially the 1-G gravitational force and the inertial G force. For example, an aircraft pulling out of a dive with a centripetal acceleration of 3 g would exert 3 G of centrifugal force; at the bottom of the dive the pilot would experience the 3-G centrifugal force in line with the 1-G gravitational force, for a total of 4 G directed toward the floor of the aircraft. If the pilot could continue his circular flight path at a constant airspeed, the G force experienced at the top of the loop would be 2 G, since the 1-G gravitational force would subtract from the 3-G inertial force. Another common example of the addition of gravitational G force and inertial G force occurs during application of power on takeoff or on a missed approach. If the forward acceleration is 1 g (32.2 ft/sec^2), then the inertial force is 1 G directed toward the tail of the aircraft; the inertial force adds vectorially to the 1-G force of gravity, directed downward, to provide a resultant gravitoinertial force of 1.414 G pointing 45^o down from the aft direction.

Just as inertial forces oppose acceleratory forces, so do <u>inertial torques</u> oppose acceleratory torques. No convenient derived unit exists, however, for measuring inertial torque; specifically, there is no such thing as angular G.

To complete this discussion of linear and angular motion, the concepts of <u>momentum</u> and <u>impulse</u> are introduced. <u>Linear momentum</u> is the product of mass and linear velocity, mv; <u>angular momentum</u> is the product of rotational inertia and angular velocity, $J\omega$. Momentum is a quantity which a translating or rotating body conserves; i.e., a body cannot gain or lose momentum unless it is acted upon by a force or torque. A <u>translational impulse</u> is the product of force, F, and the time over which the force acts on a body, Δt, and is equal to the change in linear momentum imparted to the body. Thus

$$F \Delta t = mv_2 - mv_1$$

where v_1 = initial linear velocity

and v_2 = final linear velocity.

When dealing with angular motion, one defines a rotational impulse to be the product of torque, M, and the time over which it acts, Δt. A rotational impulse is equal to the change in angular momentum; thus

$$M \Delta t = J\omega_2 - J\omega_1$$

where ω_1 = initial angular velocity

and ω_2 = final angular velocity.

The above relations can be seen to have been derived from the Law of Acceleration.

$$F = ma$$

$$M = J\alpha,$$

since $$a = \frac{v_2 - v_1}{\Delta t}$$

and $$\alpha = \frac{\omega_2 - \omega_1}{\Delta t}.$$

Directions of Linear and Angular Acceleration and Reactive Forces and Torques

Because space is three-dimensional, it is necessary to describe linear motions in space by means of reference to three linear axes and angular motions by three angular axes. In aviation it is customary to speak of the longitudinal (fore-aft), lateral (right-left), and vertical (up-down) linear axes, and the roll, yaw, and pitch angular axes, as shown in Figure 2.

Most linear accelerations in present-day aircraft occur in the vertical plane defined by the longitudinal and vertical axes, since thrust is developed along the former and lift is developed along the latter axis. In

the near future, however, aircraft capable of sideward acceleration may come into the Air Force inventory, which means that accelerations in the other two spatial planes, defined by the lateral and vertical and by the lateral and longitudinal axes, will exist. A complete, three-dimensional, linear acceleration environment will then be in effect. Most angular accelerations in aircraft occur in the roll plane (perpendicular to the roll axis), and to a lesser extent in the pitch plane. Angular motion in the yaw plane is very limited in normal flying, although it occurs in substantial quantity in spins and a few other aerobatic maneuvers.

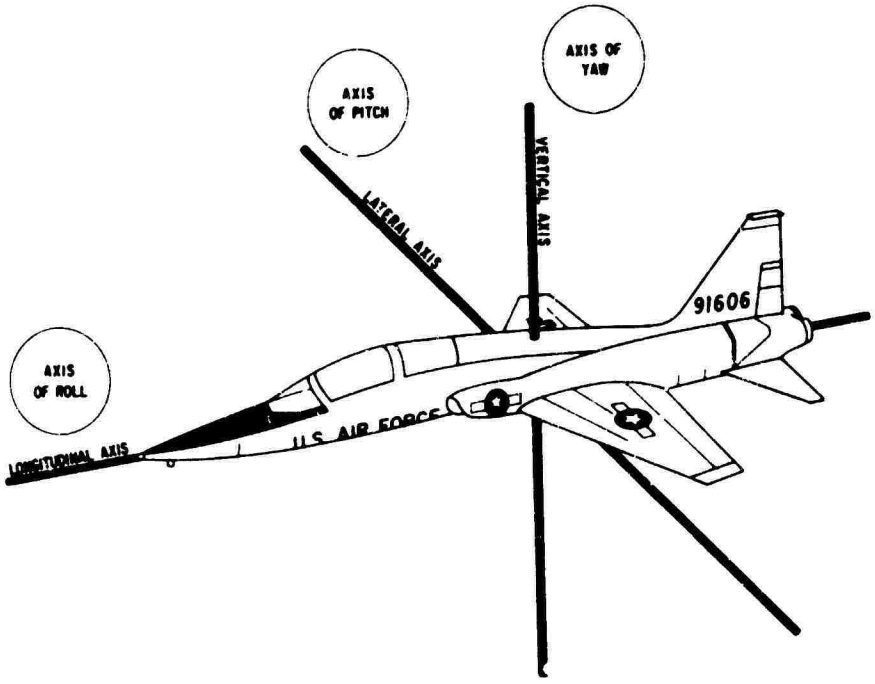


Figure 2. Aircraft axes.

A practical system for describing linear and angular accelerations acting on man is shown in Figure 3A. Note that, in this system, a linear acceleration of the type experienced on takeoff is $+a_x$, and coming to a stop after landing results in $-a_x$; radial acceleration of the type usually encountered during maneuvering is $+a_z$. The right-hand rule for describing the relations between three orthogonal axes aids recall

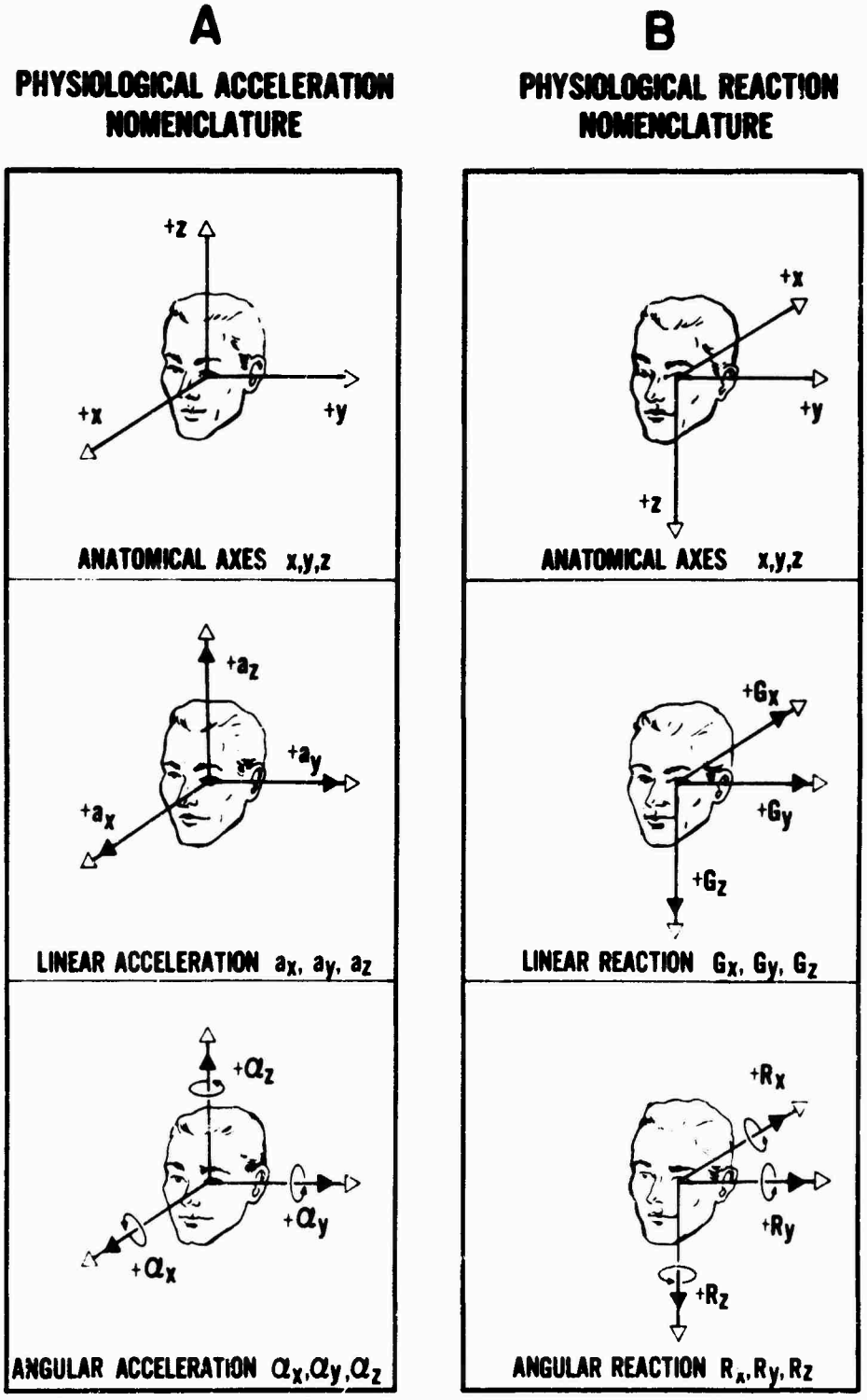


Figure 3. System for describing accelerations and inertial reactions in man. (Adapted from Hixson, Niven, and Correia, Kinematics Nomenclature for Physiological Accelerations, Naval Aerospace Medical Institute, 1966.)

of the positive directions of a_x, a_y, and a_z in this particular system: if one lets the forward-pointing index finger of the right hand represent the positive x-axis, and the left-pointing middle finger of the right hand represent the positive y-axis, then the positive z-axis is represented by the upward-pointing thumb of the right hand. Lest the reader be misled, however, into thinking that a commonly accepted coordinate system is used by physicists, engineers, acceleration physiologists, and vestibular physiologists, let that idea be dispelled now. The system proposed by Gell (1961) and adopted by the AGARD Aerospace Medical Panel utilizes another right-hand rule for establishing the vehicular coordinates: in that system, $+a_x$ is noseward acceleration, $+a_y$ is to starboard (to the right), and $+a_z$ is floorward; an inverted right hand illustrates this set of axes. AGARD employs another system devoid of symbols, signs, and subscripts, which lends itself well to describing linear accelerations acting on humans; it consists simply of headward, footward, forward, backward, right lateral, and left lateral acceleration.

The angular accelerations, α_x, α_y, and α_z, are roll, pitch, and yaw accelerations, respectively, in the system shown in Figure 3A. Note that the relation between axes is identical to that for linear accelerations. The direction of positive angular displacement, velocity, or acceleration is established by another "right-hand rule," wherein the flexed fingers of the right hand indicate the direction of angular motion corresponding to the vector represented by the extended, abducted right thumb. Thus, a roll to the right would result from $+\alpha_x$, and cessation of the roll would follow $-\alpha_x$; a pitch up would result from $-\alpha_y$ in this system. The approach taken by the AGARD Aerospace Medical Panel in describing vehicular angular accelerations is based on the same "inverted-right-hand" coordinate system that they use for describing vehicular linear accelerations; in that system a positive roll acceleration ($\dot{p}$) is to the right, a positive pitch acceleration ($\dot{q}$) is upward, and a positive yaw acceleration ($\dot{r}$) is to the right. The descriptive system used by AGARD for naming angular accelerations acting on humans employs rather gymnastic terms, including head-right cartwheeling, backward somersaulting, and right twisting.

The nomenclature for inertial forces acting in humans has somewhat more commonality than does the acceleration nomenclature. The positive directions of the axes used for describing G forces are illustrated in Figure 3B; note that the relation of these axes follows a backward, inverted right-hand rule. In the illustrated system, $+a_x$ acceleration

results in $+G_x$ inertial force, and $+a_z$ results in $+G_z$. This correspondence of polarity is not achieved on the y-axis, however, since $+a_y$ results in $-G_y$. If the direction of $+G_y$ were reversed, full polarity correspondence could be achieved between all linear accelerations and all reactive forces, and that convention has been used by some authors (notably, Dixon and Patterson); the desirability of maintaining the mathematically conventional right-hand coordinates for the reactive forces dictates the lack of correspondence on the y-axis in the illustrated system. Conveniently, the system proposed by Gell and adopted by AGARD is identical to that depicted in Figure 3B. An example of the usage of the symbolic reaction terminology would be, "The F-15 can pull $+7.33\ G_z$ until it runs out of fuel." Another very useful set of terms for describing reactive forces comprises the "eye-balls" nomenclature: in this system, the direction of the inertial reaction of the eyeballs when the head is subjected to an acceleration is used to describe the direction of the inertial force. The equivalent expressions, "eyeballs-in acceleration" and "eyeballs-in G force," leave little room for confusion about either the direction of the applied acceleratory field or the resulting gravitoinertial force environment. Table 2 summarizes the majority of terms used in describing directions of linear accelerations and gravitoinertial forces acting on man, and relates these accelerations and forces to several types of aircraft motion.

Inertial torques can be described conveniently by means of the system shown in Figure 3B, in which the right-handed set of angular reaction axes are the same as the linear reaction axes. The inertial reactive torque resulting from $+\alpha_x$ (right roll) angular acceleration is $+R_x$, and $+\alpha_z$ (left yaw) results in $+R_z$; but $+\alpha_y$ (downward pitch) results in $-R_y$, for the reason mentioned in conjunction with the discussion of the incomplete correspondence between the linear acceleration and reaction coordinate polarities. The AGARD nomenclature for reactive torques follows a convention similar to that illustrated.

It should be apparent that, to avoid becoming confused or confusing others when dealing with directions of accelerations and inertial reactions, one should carefully identify the coordinate conventions being used. Although it is preferable from a mathematical standpoint to conform to right-hand rules whenever possible, departures from such conventions may facilitate communication in some circumstances. For nonmathematical discussions, however, terminology such as that used in the "eyeballs" convention should be perfectly adequate.

TABLE 2. EQUIVALENT TERMS FOR DIRECTIONS OF LINEAR ACCELERATIONS AND GRAVITOINERTIAL FORCES

Acceleration	Gravitoinertial force	Related aircraft motions
$+a_z$ Headward	$+G_z$ Footward Positive Eyeballs-down	Level flight Coordinated turn Pull-up from dive "Inside" maneuvers
$-a_z$ Footward	$-G_z$ Headward Negative Eyeballs-up	Inverted flight Push-over into dive "Outside" maneuvers
$+a_x$ Forward	$+G_x$ Backward Positive transverse Chest-to-back Supine Eyeballs-in	Increasing forward velocity (e.g., application of afterburner) Steep climb
$-a_x$ Backward	$-G_x$ Forward Negative transverse Back-to-chest Prone Eyeballs-out	Decreasing forward velocity (e.g., application of speed brakes) Steep dive
$-a_y$ To right	$+G_y$ To left Left lateral Eyeballs-left	Left slip Left skid
$+a_y$ To left	$-G_y$ To right Right lateral Eyeballs-right	Right slip Right skid

SHORT-DURATION $+G_z$

Fighter aircraft of the 1960s and early 1970s, such as the F-4E, are limited by aerodynamic design and available power (thrust) as to the duration of increased G they are capable of developing. Consequently, during the time period that these aircraft are operational, the physiology of short-duration $+G_z$ is more important to the fighter pilot and flight surgeon than the high-sustained-G information that follows later in this review.

Cardiovascular Effects

In this instance "short-duration $+G_z$" is defined as an exposure to $<+6$ G_z for a time period varying from 3 to 15 seconds. The physiologic effects of short-duration acceleration are primarily due to the increased hydrostatic pressures developed in the vascular system. Figure 4 depicts the effect on systolic arterial pressure at +1 G_z and +6 G_z. Under conditions of +1 G_z the 30-cm fluid column from the heart (level of the third intercostal space) to the brain exerts a hydrostatic pressure of 22 mm Hg. Therefore, if systolic arterial pressure at the heart is 120 mm Hg, the arterial pressure at the base of the brain will be 98 mm Hg and 175 mm Hg at the feet. For each additional $+G_z$, the blood pressure at brain level would be reduced another 22 mm Hg, eventually producing a theoretical pressure of zero at approximately +5.5 G_z and a subatmospheric pressure of -12 mm Hg at +6 G_z. Of course, cerebral perfusion pressures of relaxed individuals during exposures to $+G_z$ cannot be accurately predicted using the aforementioned circulatory-system model because of cardiovascular compensatory adjustments. These adjustments occur approximately 6-10 sec after acceleration onset (Figure 5).

During the application of rapid-onset $+G_z$ forces ($\geq$ 1 G/sec) the increasing hydrostatic pressure produces decreasing arterial pressure at head level and blood pooling in the venous capacitance vessels below the heart. The sequence of cardiovascular events during the exposures of a subject to his $+G_z$ tolerance level is shown in Figure 6.

The decrease in blood pressure and circulating blood volume initiates a series of reflex responses of the circulatory system. Stretch receptors in both the high-pressure (elastic) system and low-pressure (capacitance) system play an important role in the maintenance of functional homeostasis.

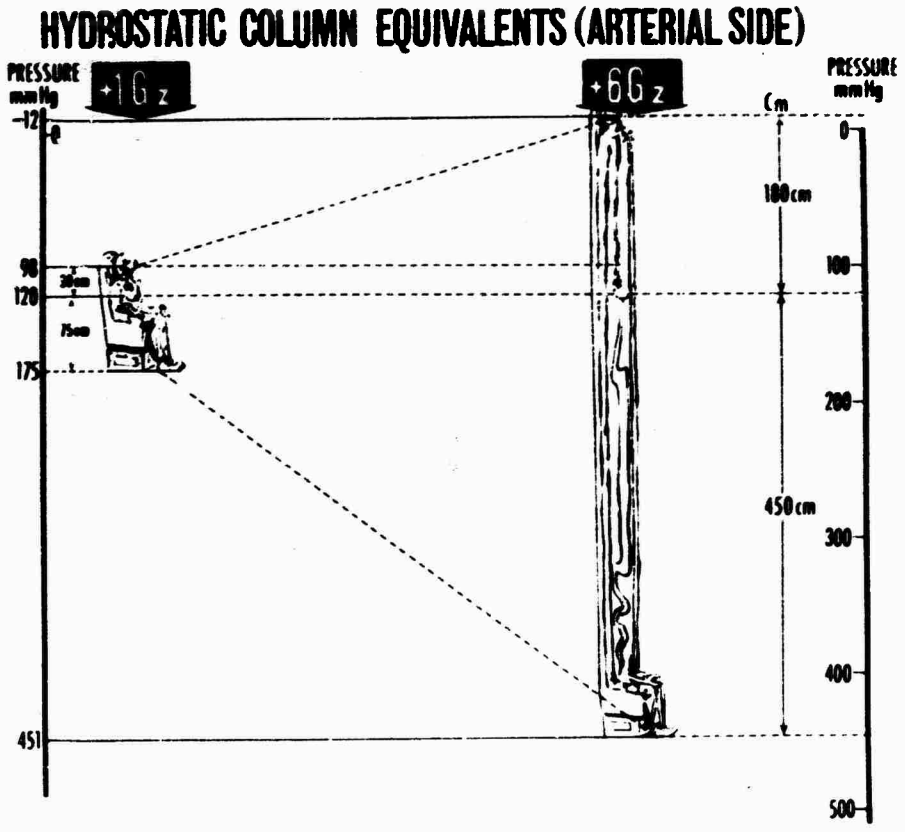


Figure 4. Hydrostatic pressure and systolic arterial pressure of a man seated in the upright position at +1 G_z and +6 G_z. (From Burton, Leverett, and Michaelson, 1974)

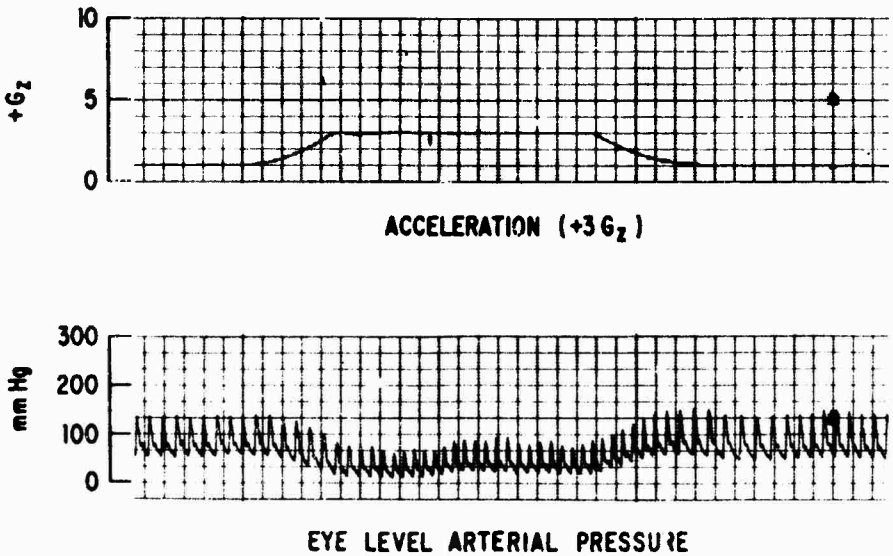


Figure 5. Blood-pressure compensation after prolonged exposure to +G_z.

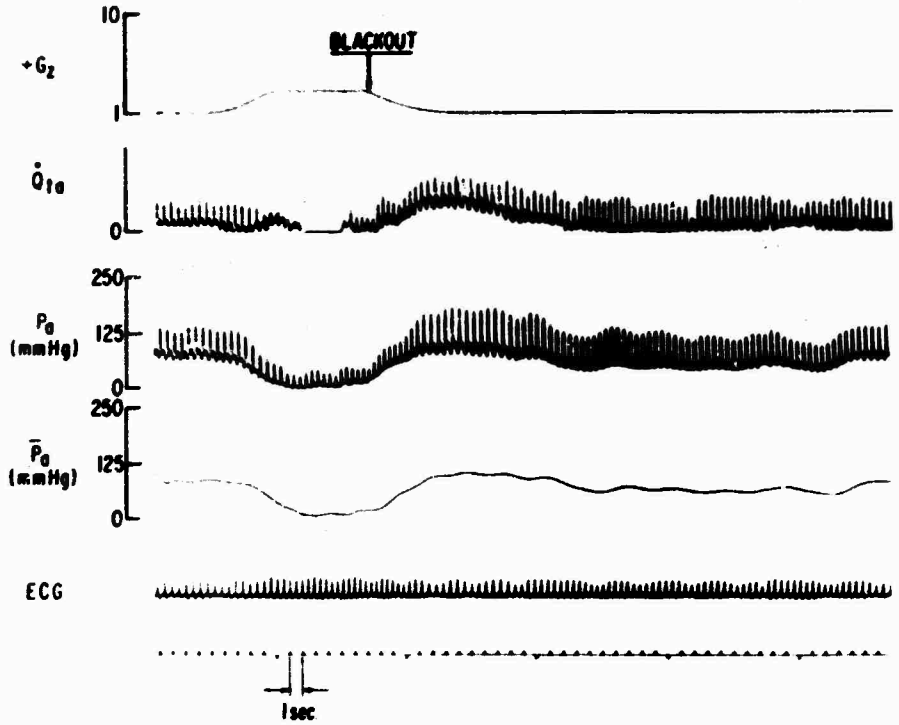


Figure 6. Cardiovascular responses in an individual exposed to his $+G_z$ tolerance level. Note that the fall in eye-level blood pressure (P_a) and mean eye-level blood pressure ($\bar{P}_a$) is accompanied by a decrease in temporal artery bloodflow velocity ($\dot{Q}_{ta}$). (From Krutz, Rositano, and Mancini, 1975)

Two areas of the high pressure system are equipped with stretch receptors: the carotid sinus (located at the bifurcation of the carotid artery in the neck) and the arch of the aorta. When the arterial pressure falls, the inhibitory discharge in these buffer nerves decreases and the brain stem reticular formation is activated producing vasoconstriction and an increased heart rate and force of contraction. Of these two high-pressure-system areas, the primary input is from the carotid sinus, where the change in hydrostatic pressure first occurs and is maximal. Arterial pressure changes at heart level during $+G_z$ are less dramatic than found above the heart and therefore the protection derived from the aortic receptors is minor.

The atrial stretch receptors perform a similar function on the low-pressure side of the cardiovascular system during $+G_z$. Very small decreases in filling volume will lead to a sharp decrease in cardioinhibitory atrial receptor afferent discharge rate. The fall in atrial

pressure works in synergy with the high-pressure receptors during $+G_z$ to increase heart rate and force of contraction, thereby helping to maintain cardiac output. Sympathetic outflow down the renal nerves promptly increases renin release and subsequently increases aldosterone secretion and fluid retention. In addition, vagal afferents to the hypothalamus increase the secretion of antidiuretic hormone (ADH) also leading to fluid retention. Finally, the decreased stretch of the atrial volume receptors mediates the sympathetic discharge which shifts renal blood from the outer cortex to the medulla, thus promoting sodium resorption. These latter three low-pressure-system responses play a progressively more important role as the duration of the $+G_z$ stress is increased.

Retinal and Cerebral Effects

In addition to the reflex cardiovascular compensatory mechanisms the cerebral circulation is protected by (1) a fall in jugular venous pressure (Figure 7) which helps maintain the arteriovenous pressure differential and (2) the decrease in cerebrospinal fluid pressure which reduces intracranial resistance to blood flow.

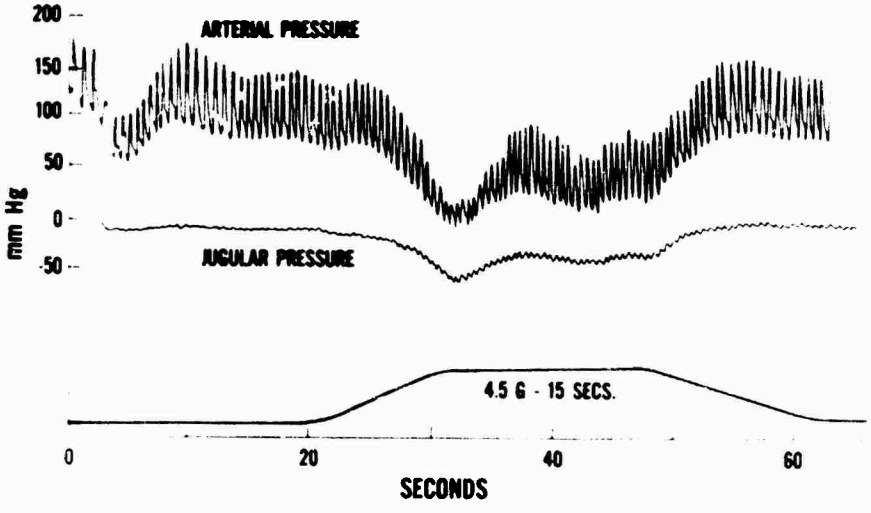


Figure 7. The effect of $+G_z$ upon cerebral arterial and jugular venous pressure. Note that a high arteriovenous pressure gradient is maintained because of a marked fall in jugular venous pressure during acceleration. (From Henry, Gauer, Kety, and Kramer, 1951)

The hemodynamics of the retinal circulation are generally considered analogous to those of the cerebral circulation. However, during $+G_z$ exposures, vision fails before unconsciousness occurs. This is attributed to the 20-mm Hg intraocular pressure which must be overcome for retinal perfusion to occur. Changes in blood flow in the fundus oculi during $+G_z$ have been correlated with subjective visual responses using direct ophthalmoscopy (Table 3). Retinal photography and fluorescence angiography have been used to show the gradual collapse of the retinal circulation. Figure 8 shows the correlation between eye-level blood pressure changes and the retinal circulation during $+G_z$.

TABLE 3. CORRELATION BETWEEN SUBJECTIVE VISUAL RESPONSES AND FUNDUS OCULI CHANGES AS DETERMINED BY OPHTHALMOSCOPY. (FROM DUANE, 1954).

Stage	Subjective response	Objective response
I	Loss of peripheral vision	Arteriolar pulsation--i.e., recurrent exsanguination
II	Blackout	Arteriolar exsanguination and collapse
III	Return of central and peripheral vision	Return of arteriolar pulsation and temporary venous distension

Visual failure is a continuum from loss of peripheral vision (grey-out) to total loss of vision (blackout) to unconsciousness. Peripheral vision is lost first because the retinal circulation is an end-artery system, with vessels of decreasing diameter extending from the central retinal artery to the periphery. Therefore, when central pressure falls, collapse will occur first in the periphery of the retina.

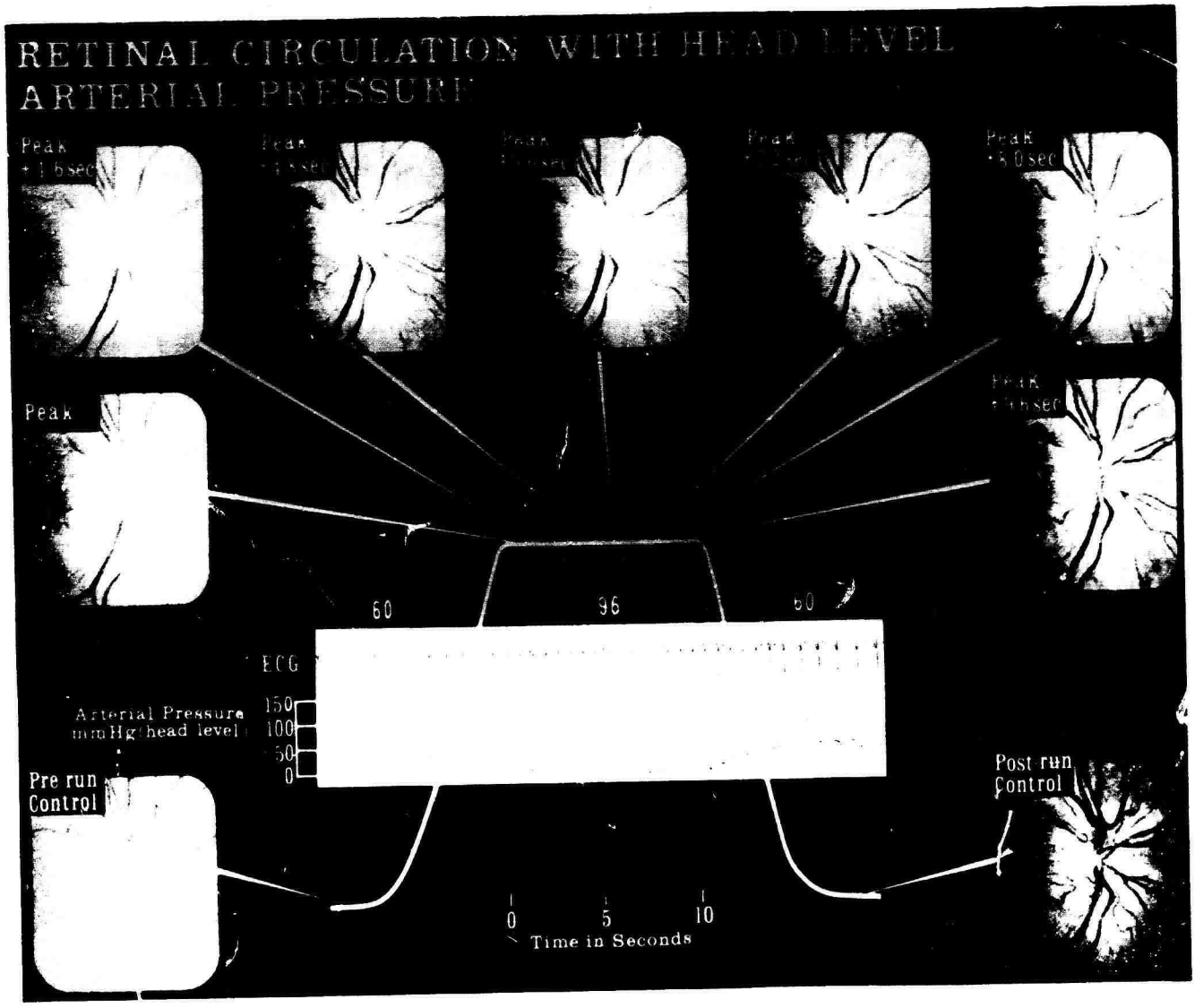


Figure 8. Changes in retinal circulation and eye-level blood pressure during blackout on the human centrifuge. (From Leverett and Newsom, 1971)

Thresholds of visual impairment resulting from $+G_z$ exposure vary widely among individuals. Table 4 displays the range of differences in individual tolerance obtained from 1,000 Naval aviation cadets who were relaxed and unprotected during the study. Blackout in normal relaxed subjects is primarily dependent on the duration and rate of onset of $+G_z$ (Figure 9). Convulsions with characteristic EEG changes often accompany unconsciousness, having been found in 52% of 230 subjects. The additive effects of stagnant and hypoxic hypoxia (from impaired respiration) are probably responsible for the long recovery period, which may take as long as one minute, following cessation of the acceleration. Furthermore, a period of disorientation may also be present for some time after consciousness is regained. Frequently, the subject or aircrew member is unaware of unconsciousness having occurred during acceleration exposure. The pilot and flight surgeon therefore should be aware of the events leading to unconsciousness so that this state can be avoided at all costs.

TABLE 4. RANGE OF VISUAL THRESHOLDS IN RELATION TO $+G_z$ TOLERANCE (FROM COCHRAN, GARD, AND NORSWORTHY, 1954)

Symptom	Mean threshold	Standard deviation	Range
Loss of peripheral vision (grayout)	4.1 G	± 0.7 G	2.2 to 7.1 G
Blackout	4.7 G	± 0.8 G	2.7 to 7.8 G
Unconsciousness	5.4 G	± 0.9 G	3.0 to 8.4 G

The range of G forces required to produce the effects listed displays the wide difference in individual tolerance encountered in a large group of people. These values were obtained from centrifuge studies using a group of 1,000 Naval aviation cadets as subjects who were relaxed and unprotected during the study.

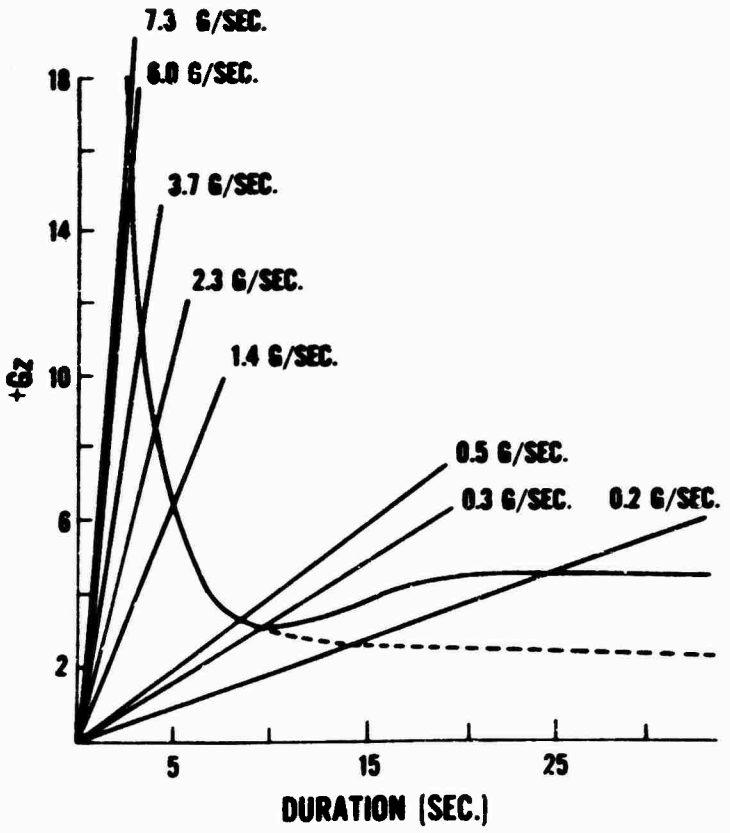


Figure 9. Human acceleration intensity/duration blackout tolerance curves compared with various acceleration onset rates. Note that acceleration-intensity tolerance is a function of both duration and onset rates. (From Stoll, 1956)

Respiratory Effects

At +1 G_z a pressure gradient exists between the apex and base of the lungs in the upright individual. At increased G loads the ventilation/perfusion ratio ($\dot{V}_A$:$\dot{Q}$) is increased in the upper portion of the lung, whereas in the lower lung this ratio is decreased. This change in $\dot{V}_A$:$\dot{Q}$ produces a progressive decline in arterial oxygenation during prolonged $+G_z$ exposures (Figure 10). This subject will be considered in greater detail, later in this review, in the section titled "High Sustained G_z."

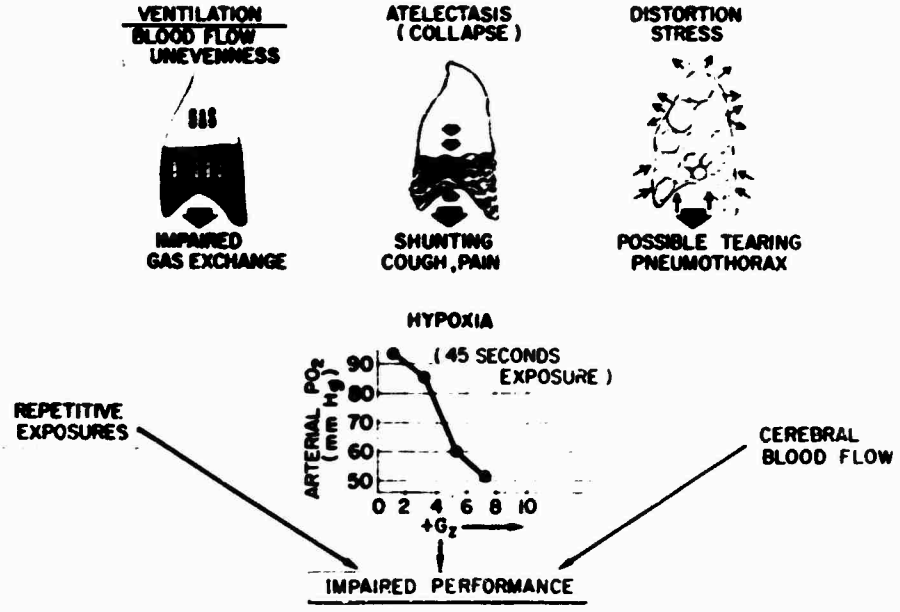
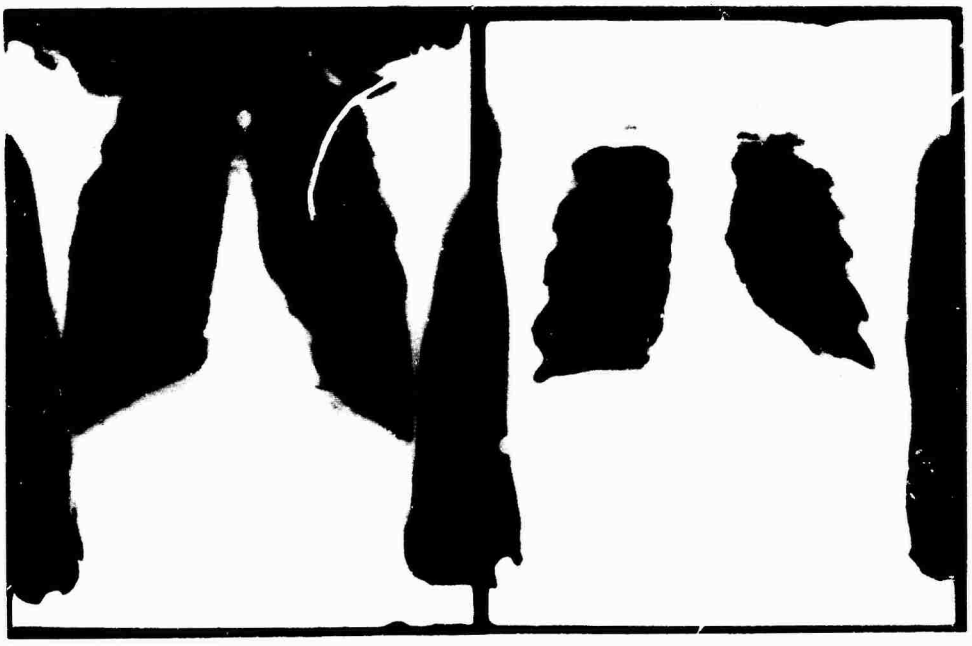


Figure 10. Effects of $+G_z$ on the lung.

During short-duration $+G_z$ exposure, with the upward displacement of the diaphragm by an inflated anti-G suit (Figure 11) combined with breathing 100% oxygen, compression and absorption atelectasis occur. In this regard, pilots of high-performance aircraft (who are required to breathe 100% oxygen because of regulator design) frequently develop atelectasis. Interestingly, this atelectatic condition is more pronounced in pilots who smoke cigarettes.

Tolerance

A measure of man's capability at $+G_z$ is usually determined while the subject is relaxed and is quantified by the G level at which specific physiologic systems are significantly altered. The most common criteria are visual changes (various stages of light loss experienced by the subject): peripheral light loss (PLL), central light dim (CLD), and central light loss (CLL). However, other criteria may be used (such as auditory phenomena, cardiorespiratory responses, and consciousness). The light-loss criterion, although rather subjective, has the advantage of simplicity. Since hearing is the last sensory modality remaining just



+1 G_z +7 G_z

Figure 11. Effect of anti-G-suit inflation on lung and heart position during $+G_z$.

prior to unconsciousness intervening during $+G_z$, it is not a recommended endpoint. Human tolerance to $+G_z$ in relaxed individuals without anti-G suit protection can vary from 3.6 G - 4.1 G, using blackout or loss of vision (CLL) as an endpoint.

Evaluation of Low G Tolerance in Aircrew Members

Some aircrew members black out or lose consciousness at unusually low G levels for reasons that cannot readily be detected by the flight surgeon. Usually the pilot with low-G-tolerance symptoms is discovered by his instructor pilot during the aerobatic phase of undergraduate pilot training. The flight surgeon should be aware of the use of the human centrifuge at the USAF School of Aerospace Medicine as an adjunct to the physical examination of these individuals. The centrifuge can be used to impose a controlled G stress on the pilot (or trainee), using

variable onset rates of G, thus allowing a physician monitor to determine with a high degree of accuracy the pilot's relaxed and protected (straining with or without an anti-G suit) blackout levels. In many instances it has been found that the individual is performing an improper straining maneuver, and this is the cause of his abnormally low blackout level. An explanation of both the proper and improper straining maneuvers is found later in this review in the section titled "M-1 and L-1 Maneuvers." Using the closed-circuit video tape recording/playback facility associated with the human centrifuge at the School of Aerospace Medicine, the pilot can observe his prior centrifuge runs in which the performance of a straining maneuver was inadequate to maintain vision. He then learns the proper protective measures and, most important, learns proper pacing of his respiratory cycle while straining during prolonged G exposure. If the individual being examined does not demonstrate an increase in his tolerance of at least 1.5 G (compared to his relaxed blackout level) when these adjunct protective measures are used, then some underlying pathologic condition should be suspected.

Protection Against the Effects of Short-Duration $+G_z$

Body Position--The simplest and most effective means of increasing tolerance to short-term $+G_z$ is to change the position of the body's long axis relative to the inertial force. This reduces the apparent height of the hydrostatic column of blood extending from the heart to the head. Figure 12 demonstrates the reduction in the $+G_z$ vector obtained by backward tilting. Interestingly, relaxed $+G_z$ tolerance is not significantly increased until a 45° tilt from the vertical is obtained (Figure 13). Prototypes of the new lightweight fighter (LWF) are including a tilt-back seat in hopes of increasing G tolerance.

M-1 and L-1 Maneuvers--The M-1 and L-1 maneuvers are straining techniques used by pilots to improve G tolerance. Pilots commonly refer to the M-1 maneuver as the "grunt" maneuver since it approximates the physical effort required to lift a heavy weight. The M-1 maneuver consists of pulling the head down between the shoulders, slowly and forcefully exhaling through a partially closed glottis, and simultaneously tensing all skeletal muscles. Pulling the head downward gives some degree of postural protection (shortens the vertical head-heart distance); intrathoracic pressure is increased by strong muscular expiratory efforts against a partially closed glottis; and the contraction of abdominal and

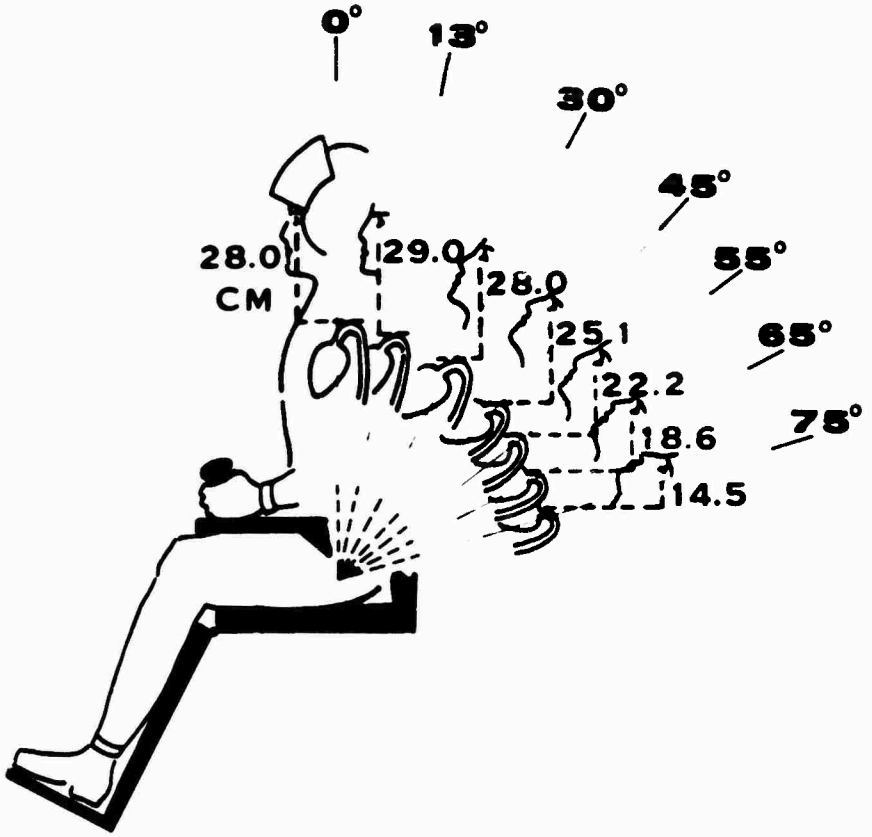


Figure 12. Decreased vertical heart-to-eye distances obtained by tilting backward. (From Burns, 1975)

peripheral muscles raises the diaphragm and externally compresses capacitance vessels. For long-duration G exposures, the maneuver must be repeated every 4 to 5 seconds. When properly executed, the exhalation phase of the M-1 results in an intrathoracic pressure of 50 to 100 mm Hg, which raises the arterial blood pressure at head level and thereby increases $+G_z$ tolerance at least 1.5 G. The inspiratory phase of the M-1 maneuver must be a fast "gasp," to be followed immediately by the exhalation phase, since mean blood pressure falls to approximately zero during inspiration (Figure 14).

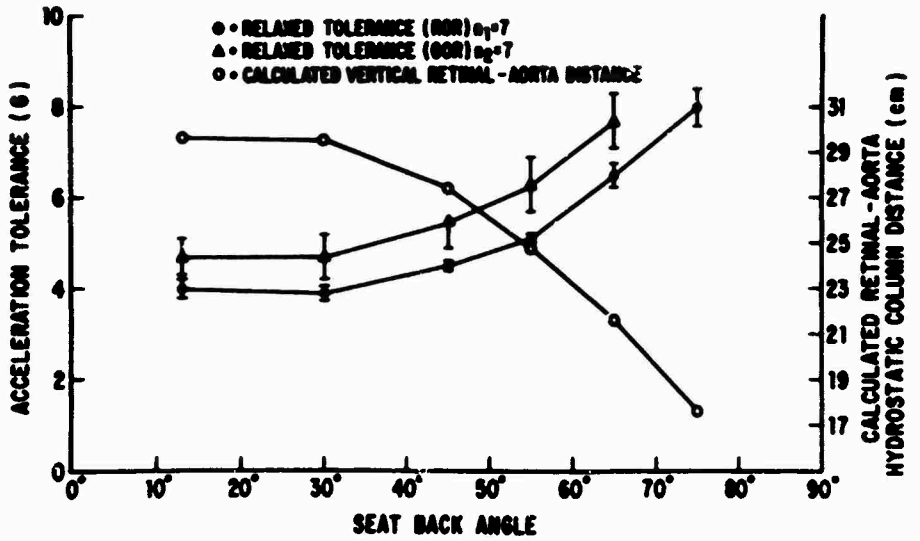


Figure 13. Increased $+G_z$ tolerances obtained by tilting backward during both rapid-onset runs (ROR, 1 G/sec) and gradual-onset runs (GOR, 0.1 G/sec). Note that no significant increase in tolerance is seen until a seat-back angle of 45° is attained. (From Burns, 1975)

The L-1 maneuver is similar to the M-1 maneuver except the aircrew member forcefully attempts to exhale against a completely closed glottis while tensing all peripheral skeletal muscles. Using either maneuver the pilot obtains equal protection, i.e., 1.5 G greater than relaxed blackout level with or without the anti-G suit: However, it is important to note (a word of caution) that forcefully exhaling against a closed glottis without vigorous skeletal muscular tensing (Valsalva maneuver) can reduce $+G_z$ tolerance and lead to an episode of unconsciousness at extremely low G levels. Therefore, instruction on the proper method of performing these straining maneuvers is essential.

Anti-G Suits--Extensive research has been conducted in designing mechanisms to externally support the cardiovascular system. Different techniques which have been used vary from merely taping the lower part of the body and limbs, as the Japanese did in World War II, to complicated pulsating pneumatic systems. The theoretical basis of all anti-G

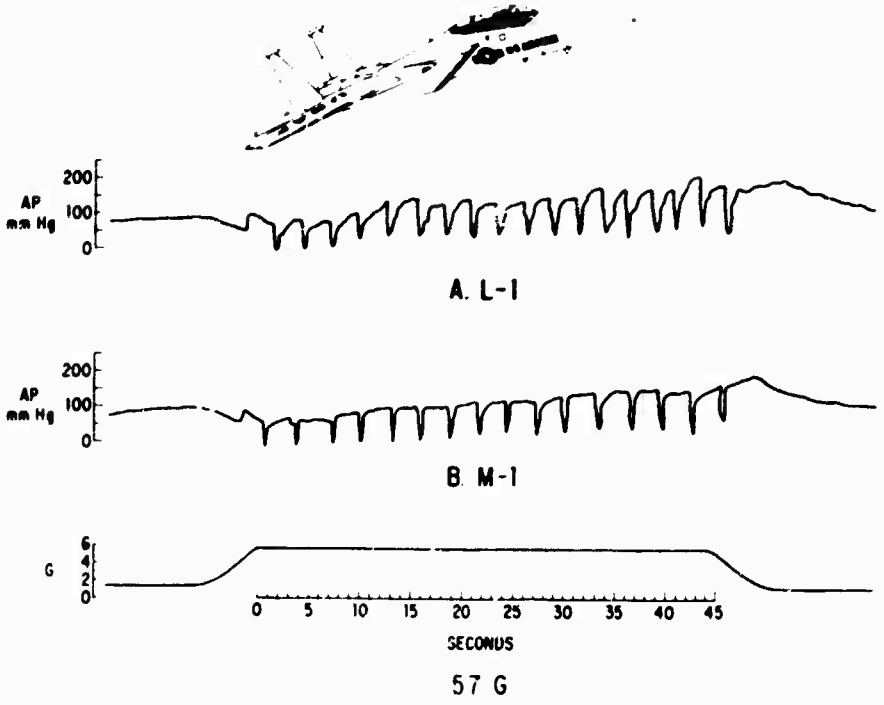


Figure 14. Mean eye-level blood-pressure changes during $+G_z$ while performing the M-1 and L-1 maneuvers. Mean blood pressure falls to zero during the inspiratory phase of either maneuver.

garments is to supply external pressure to the lower part (below heart level) of the body. Pressure on the abdomen prevents descent of the diaphragm which helps to maintain the same heart-to-brain distance, while compression of the legs prevents pooling of blood and assists in maintaining cardiac output. The Air Force anti-G garment consists of 5 interconnected pneumatic bladders which support the abdomen and legs (Figure 15). The system is controlled by a valve which regulates the amount of air entering the anti-G suit bladders. The valve is actuated at +2 G_z and thereafter increases anti-G suit pressure at the rate of 1.5 psi/G to a maximum of 10.5 psi. The anti-G suit gives about 1.5 G increased tolerance above normal relaxed values (Figure 16).

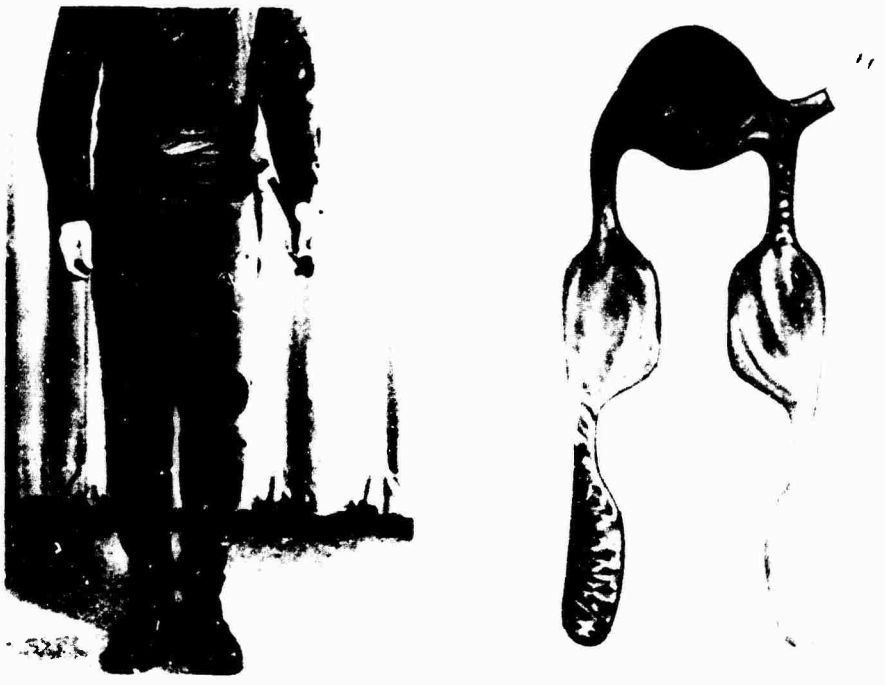


Figure 15. Standard 5-bladder Air Force anti-G suit.

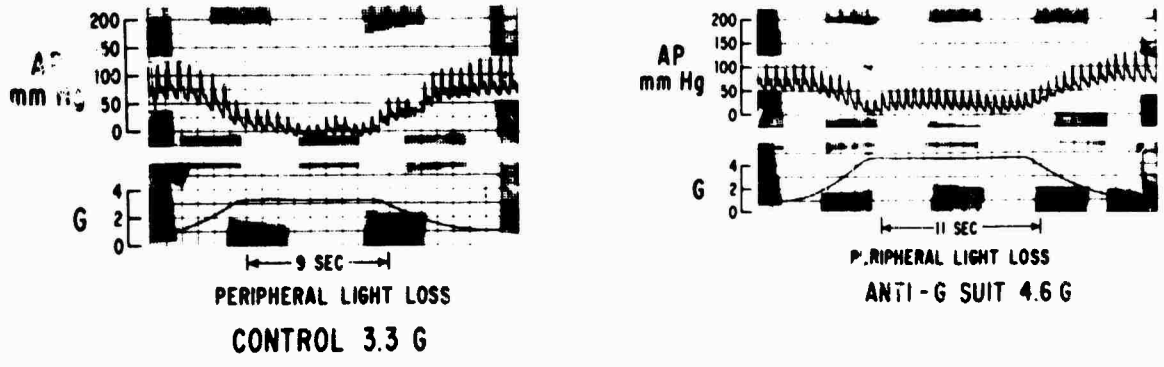


Figure 16. Increase in $+G_Z$ tolerance afforded by a standard 5-bladder anti-G suit (relaxed subject). (From Shubrooks and Leverett, 1973)

Carbon Dioxide--Inspired carbon dioxide locally dilates cerebral vessels, centrally constricts peripheral blood vessels, and shifts the oxyhemoglobin saturation curve to the right, thus improving the supply of oxygen available to the cerebral tissue. Breathing an air mixture containing 5.2% or 7.9% CO_2 increases $+G_z$ tolerance by 0.5 G and 0.9 G respectively. These values were obtained from relaxed subjects who did not wear anti-G suits.

Factors Decreasing Tolerance to $+G_z$

Any factor that reduces the overall efficiency of the body, especially if it reduces the reserve of the circulatory system, causes some reduction in man's tolerance to G. The following list is composed of some specific entities associated with lowered G tolerance:

(1) Chronic and acute hypotension
(2) Hot temperature (increased body heat load)
(3) All types of hypoxia
(4) Hypoglycemia
(5) Environmental or self-imposed stress factors. The self-imposed stresses cause G tolerance to vary on a day-by-day basis.
(6) Dehydration
(7) Any infectious process leading to a febrile condition.

Also, the ability to track a target at G is significantly decreased after alcohol ingestion (Figure 17). The combination of blood alcohol and $+G_z$ resulted in a potentiated reduction in performance. Several physical abnormalities (such as varicose veins, hemorrhoids, hernia, high myopia, and glaucoma) also exist which should preclude the individual from increased $+G_z$ exposures.

HIGH SUSTAINED $+G_z$

High sustained $+G_z$ (HSG) is defined as exposures to $+6$ G_z and greater for periods longer than 15 sec. New fighter aircraft such as the F-15 Eagle and prototypes of the Lightweight Fighter are capable of producing HSG which approach or may even exceed man's tolerance. In fact, man's tolerance to $+G_z$ may limit the performance of the aircraft. Consequently, it is extremely important that pilots of these

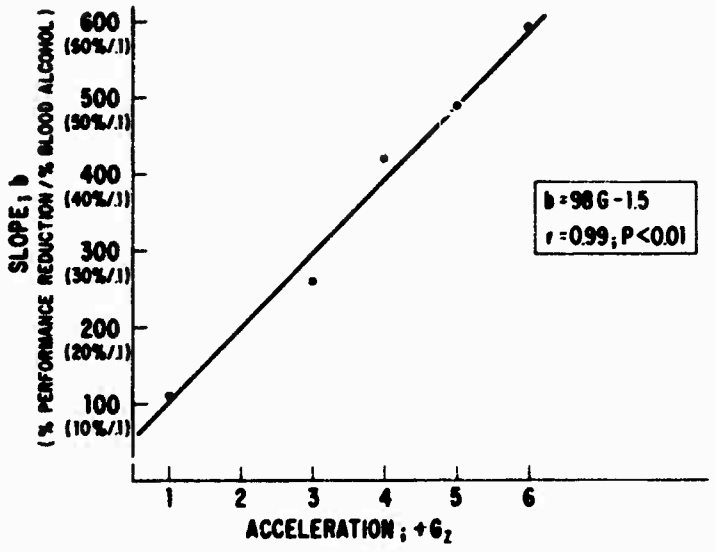


Figure 17. The relationship of percent reduction in task performance per percent blood alcohol concentration (b) with acceleration exposure. (From Burton and Jaggars, 1974)

high performance aircraft understand the physiologic effects of HSG and all available anti-G techniques.

Tolerance

In a recent study 9 out of 14 experimental subjects were able to endure +9 G_z for 45 seconds without loss of vision. All subjects were able to withstand +7.5 G_z for 45 seconds. In order to attain these levels a comprehensive training program was initiated which included instruction in the proper performance of the L-1/M-1 straining maneuver and weekly increases in +G_z level in increments of 0.5 G (Figure 18). A standard U.S. Air Force 5-bladder anti-G suit was worn. Some subjects felt they could achieve still higher +G_z levels than the established experiment maximum. In a later study, highly trained subjects were exposed to a maximum of +8 G_z for 60 seconds. All experimental subjects tolerated +6 G_z for 60 seconds; however, several subjects failed to complete the entire +8 G_z run principally because of fatigue and/or loss of vision.

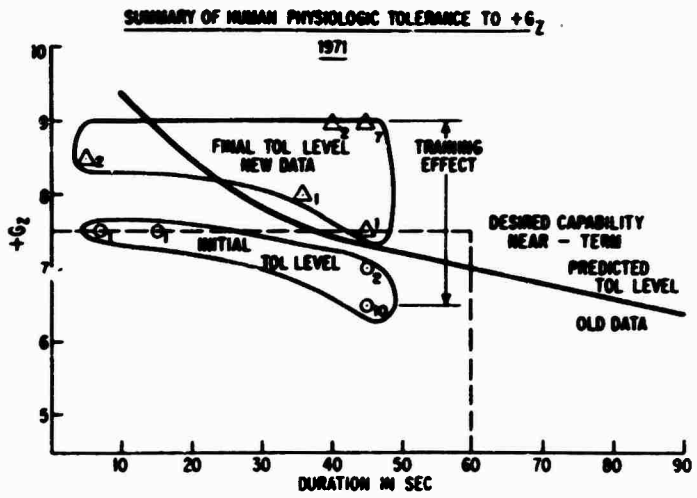


Figure 18. Effect of a comprehensive training program on raising subjects' tolerances to high sustained $+G_z$.

Cardiovascular Effects

Premature ventricular contractions (PVCs) frequently occur in subjects exposed to HSG. (Because of ECG artifacts induced by muscular contraction at HSG, other ECG changes are difficult to evaluate.) The occurrence of PVCs was twice as frequent at +9 G_z when compared to +7 G_z in subjects who tolerated both G levels, and PVCs occasionally occurred in runs of 4 to 7. Although cardiac rate and rhythm changes associated with HSG are considered not "serious," additional stresses could induce more important rhythm disturbances.

Heart rate increases with the level of HSG until +7 G_z is reached, when it appears that further increases in G cause little or no increase in heart rate. Much of this heart rate increase occurs prior to G onset (psychologic in origin), and the effort expended during performance of the M-1 contributes significantly to the high heart rate; e.g., the heart rates for persons performing a maximum M-1 at +1 G_z are the same as those found during exposures to +6 G_z.

Occasionally, during exposure to HSG, a decrease in heart rate occurs. This heart rate response is called "high-G bradycardia" and may be attributed to a reflex baroreceptor response to the high arterial pressure

resulting from the M-1 maneuver. However, the occurrence of a Bezold-Jarisch type reflex resulting from decreased ventricular filling may be a contributing factor.

Pulmonary Function

As might be expected, sustained exposure to $+G_z$, where the subject has large changes in regional $\dot{V}_A:\dot{Q}$ ratios, results in a rapidly developing hypoxemia (Figure 19). The effect of various levels of HSG on P_{aO_2} is shown in Figure 20. In the lower portion of the lung the $\dot{V}_A:\dot{Q}$ ratio is greatly reduced and both physiologic and anatomic right-to-left shunting occurs. Absorption regional atelectasis occurs post-HSG even in persons breathing air (without additional oxygen). Of course this atelectatic condition would be magnified at G if 100% oxygen were breathed. Also, the additional oxygen would be of limited value during G because much of HSG hypoxemia results from anatomic shunting.

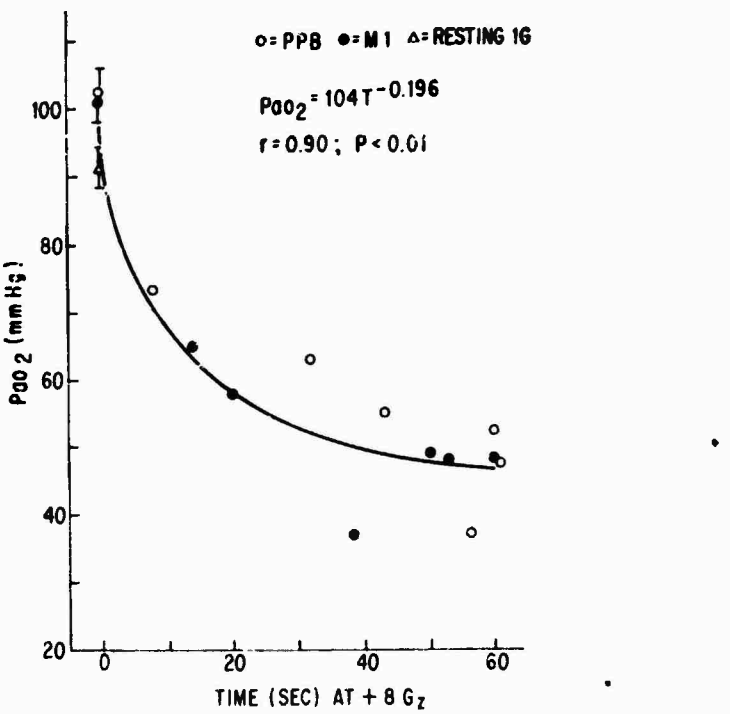


Figure 19. The effect of exposure time at +8 G_z on P_{aO_2} in subjects performing the M-1 maneuver or while positive-pressure breathing. (From Burton, Leverett, and Michaelson, 1974)

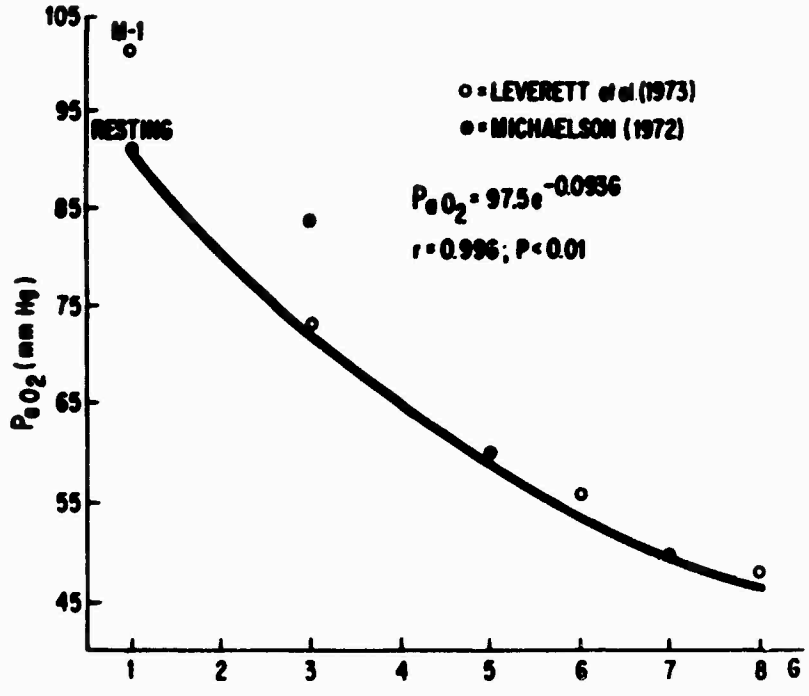


Figure 20. Changes in P_{aO_2} associated with various levels of HSG. (From Burton, Leverett, and Michaelson, 1974)

Respiratory rates increase with increasing levels of HSG. However, above 5 G tidal volume (V_T) remains essentially constant--apparently a function of abdominal compression by the inflating anti-G suit.

Cigarette Smoking

Eleven volunteer subjects were exposed to 7-9 $+G_z$ for 45 seconds and vital capacity (VC) was determined 60-90 sec post-G. Four subjects were cigarette smokers ($\geq$ one pack/day); seven were nonsmokers. The four smokers had a significant decrease in VC after HSG exposure, when compared to their prerun control values and to the VC of the nonsmokers.

Methods to Combat the Effects of HSG

Anti-G suits--A standard 5-bladder U.S. Air Force anti-G suit (CSU-12/P, CSU-3/P) was compared with the RAF mini-anti-G suit, which is essentially the same as the USAF suit only without the calf bladders. Tolerance to HSG was the same with both suits; however, subjects reported severe calf pain with the mini-suit. More recently the pneumatic lever anti-G suit (PLS) (Figure 21), a standard 5-bladder anti-G suit, and a lower body full-bladder anti-G suit (LBFB) (Figure 22), have been used experimentally at HSG. The anti-G protective features of the PLS and LBFB are similar in that both suits afford uniform counterpressure to the abdomen and entire leg region. Results indicate that significantly greater protection (using change in heart rate during HSG as the criterion of G protection) is afforded to subjects exposed to +6 G_z for 60 seconds wearing either the PLS or LBFB, when compared to the current operational 5-bladder cut-away anti-G suit.

Anti-G Valves--The inflation rate of current anti-G valves has remained unchanged since the 1940s; i.e., 1.5 psi/G above 2 G. Until recently this inflation schedule has been adequate to provide the increase in G tolerance required in operational aircraft. In order to cope with the high G forces and their rapid onset rates as generated by newly developed aircraft, several types of anti-G valves with variable inflation schedules are being developed to arrive at an optimum anti-G suit inflation schedule. Somewhat akin to rapid inflation rates, preacceleration inflation of a standard 5-bladder suit has been shown to give greater $+G_z$ tolerance than found with standard inflation methods. It appears, therefore, that a more rapid inflation rate at the onset of $+G_z$, when the cardiovascular and pulmonary systems are most rapidly affected, will increase man's ability to withstand HSG.

Positive Pressure Breathing (PPB)--The relative effectiveness of PPB as an anti-G technique has recently been compared to the M-1 maneuver at HSG (+3, 6, and 8 G_z for 60 seconds) while subjects were wearing a standard 5-bladder anti-G suit. PPB was found to be as effective as the M-1 maneuver regarding tolerable time at +6 and +8 G_z. The majority of the subjects preferred PPB to the M-1 because of less fatigue associated with PPB.

Figure 23 shows esophageal pressures (which indicate changes in intrathoracic pressure and straining effort expended) and eye-level blood pressures in three subjects exposed to +8 G_z and using an anti-G suit and either PPB (30 mm Hg) or the M-1.

Figure 21. Pneumatic lever anti-G suit (PLS).

Figure 22. Lower body full-bladder anti-G suit (LBFB).

It can be seen that in these three subjects no consistent differences exist between arterial pressure responses and the anti-G method used; although an earlier HSG study, which also compared the M-1 with PPB (40 mm Hg) as anti-G methods, concluded that PPB resulted in less eye-level arterial pressure fluctuations associated with the respiratory cycle.

Arterial oxygen tensions (P_{aO_2}) have been compared in subjects using PPB or the M-1 maneuver at HSG. The P_{aO_2} of subjects using PPB at +3 G_z and at +6 G_z was significantly higher than that found in the same subjects

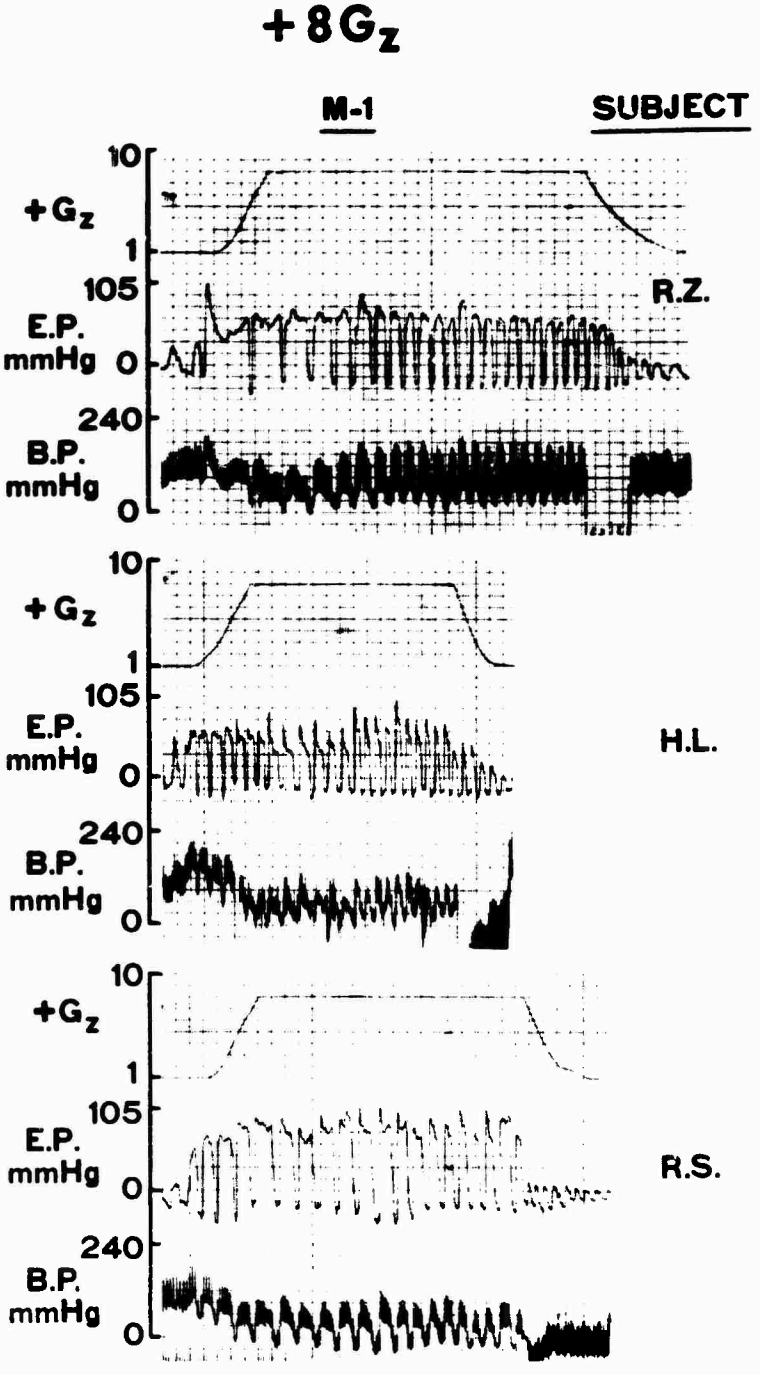


Figure 23. Physiologic changes at +8 G_z while subjects are positive-pressure breathing (PPB, 30 mm Hg), or using the M-1 maneuver. Esophageal pressure (E.P.) is used as an indication of straining required to maintain vision. Note that changes in E.P. are reflected in changes in eye-level blood pressure (B.P.). (From Burton, Leverett, and Michaelson, 1974)

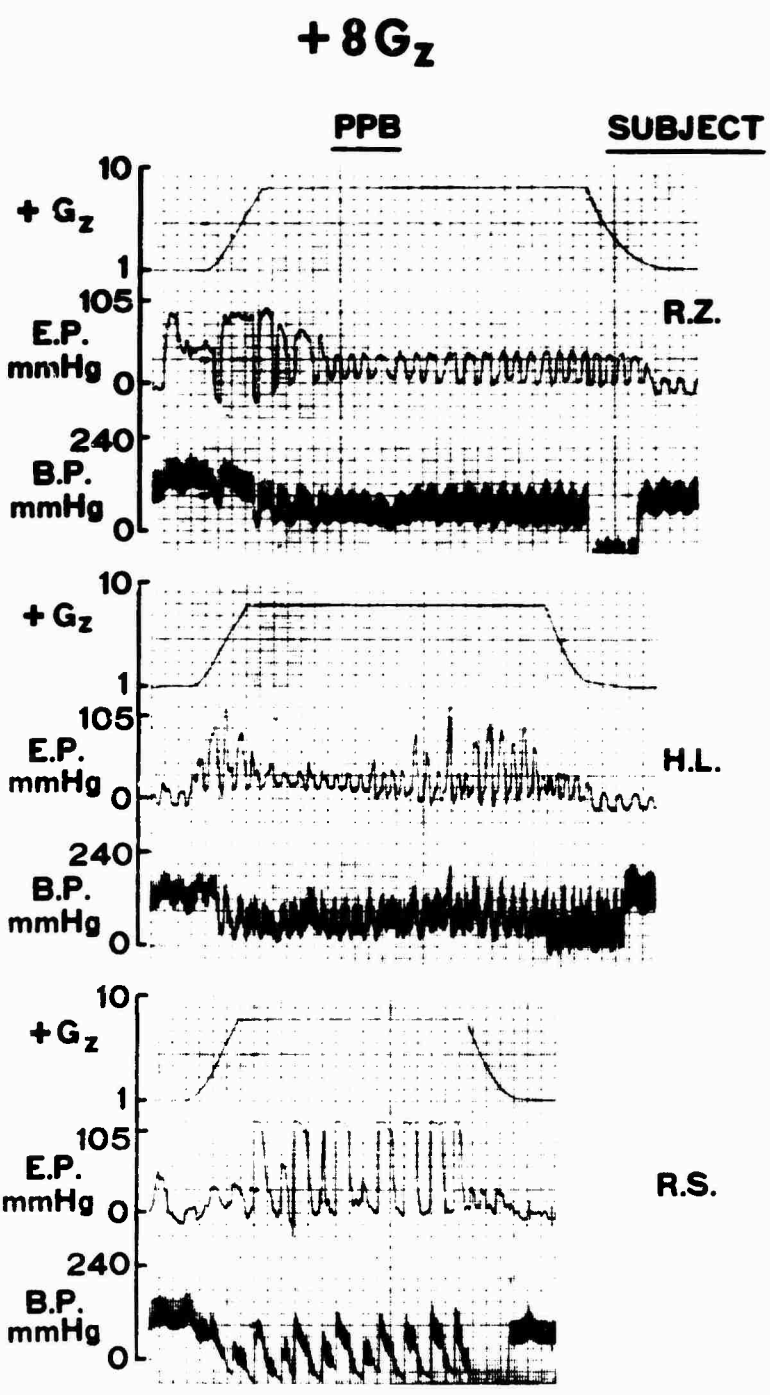


Figure 23 (continued)

using the M-1, but no difference was noted at +8 G_z (Figure 24). The relatively increased P_{aO_2} afforded by PPB at +3 and +6 G_z can be attributed to the increased O_2 requirement of the M-1 and/or a modification of the G-induced lung changes. However, the beneficial effects of PPB on lung expansion are counterbalanced by the higher G-suit pressures at +8 G_z. This may in part account for the similar values of P_{aO_2} attained from both PPB and the M-1 at +8 G_z.

These studies show that PPB with ambient air offers equivalent protection at HSG to the M-1 with less subject fatigue. It must be remembered, however, that current pressure demand oxygen regulators can only deliver 100% oxygen in the pressure settings, and the probability of an increased incidence of atelectasis associated with 100% oxygen presently prevents the use of PPB as an operational anti-G method.

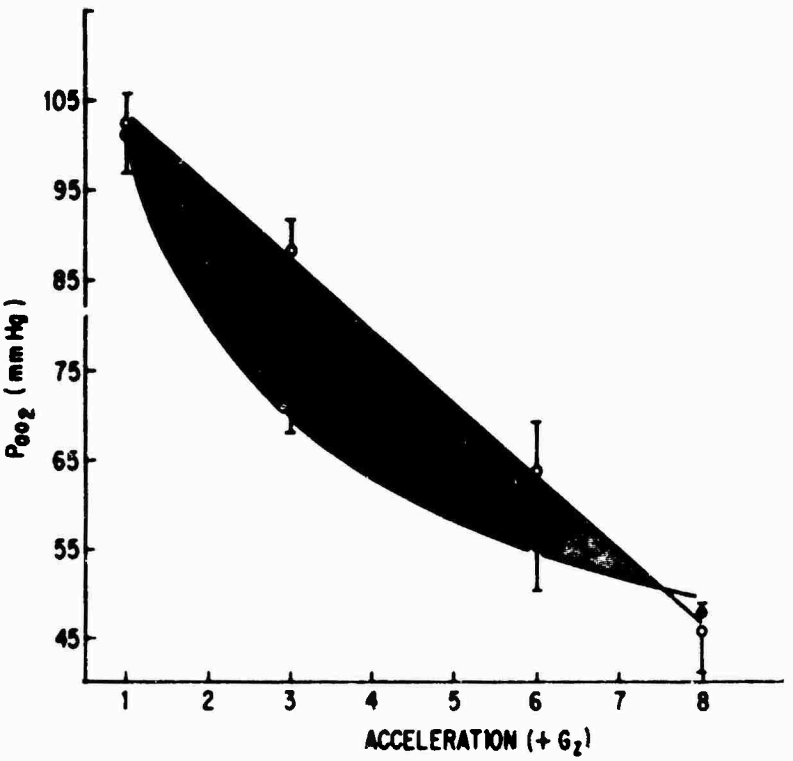


Figure 24. Changes in arterial oxygen tension (P_{aO_2}) during exposure to +G_z while using the M-1 straining maneuver (closed circles) or positive-pressure breathing (PPB) (open circles). (From Burton, Leverett, and Michaelson, 1974)

M-1 and L-1 at HSG--Many pilots prefer the L-1 to the M-1 straining maneuver because of less noise and laryngeal irritation associated with the L-1. Both maneuvers were used at HSG to evaluate their relative effectiveness. Eye-level arterial pressure responses, which fluctuated with breathing efforts, were similar for both maneuvers as shown in Figure 25. This similar response shows a gradual increase in systolic pressure as the maneuvers were repeated. The study concluded that both straining maneuvers offered an equivalent increase in HSG tolerance, with or without an anti-G suit.

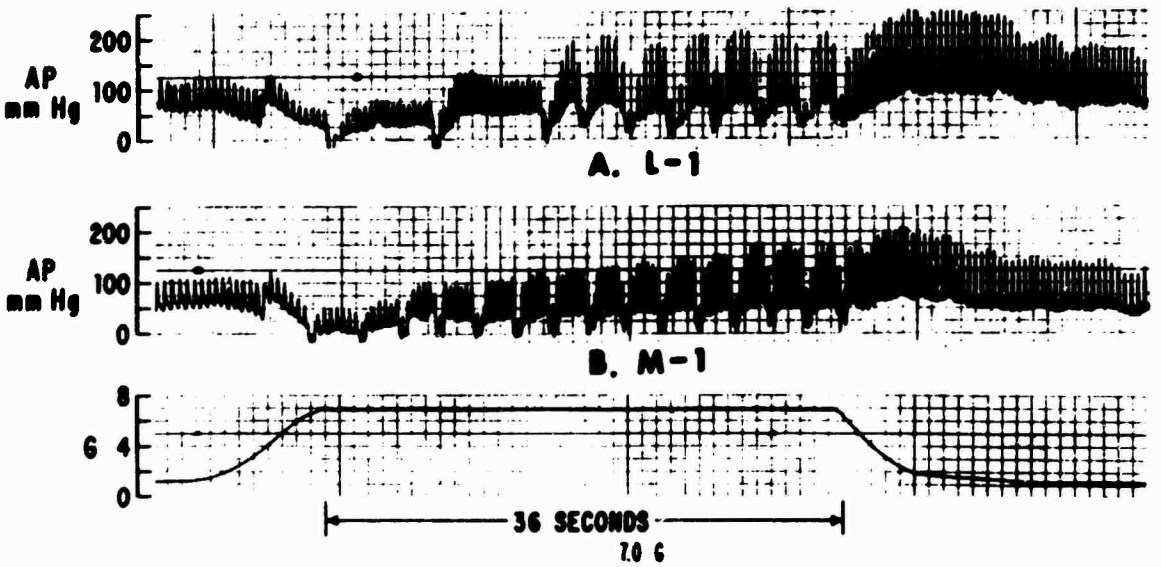


Figure 25. Eye-level blood-pressure responses while performing the M-1 and L-1 straining maneuvers at high sustained G. (From Burton, Leverett, and Michaelson, 1974)

Tilt-back Seat--Recent studies using the amplitude of the esophageal pressure (EP) as an indication of effort required to maintain vision at HSG have shown that the tilt-back seat offers significant advantages over the standard 13^{o} seat (Figure 26). Also heart rates are lower in subjects using the tilt-back seat at HSG, indicating less fatigue and cardiovascular stress. Although much of the cardiovascular stress is eased at 65^{o} tilt, large changes in the $\dot{V}_A:\dot{Q}$ ratio are present (such as found at $+G_z$) suggesting that hypoxemia may be the limiting physiologic factor in pilots using these supinating seats.

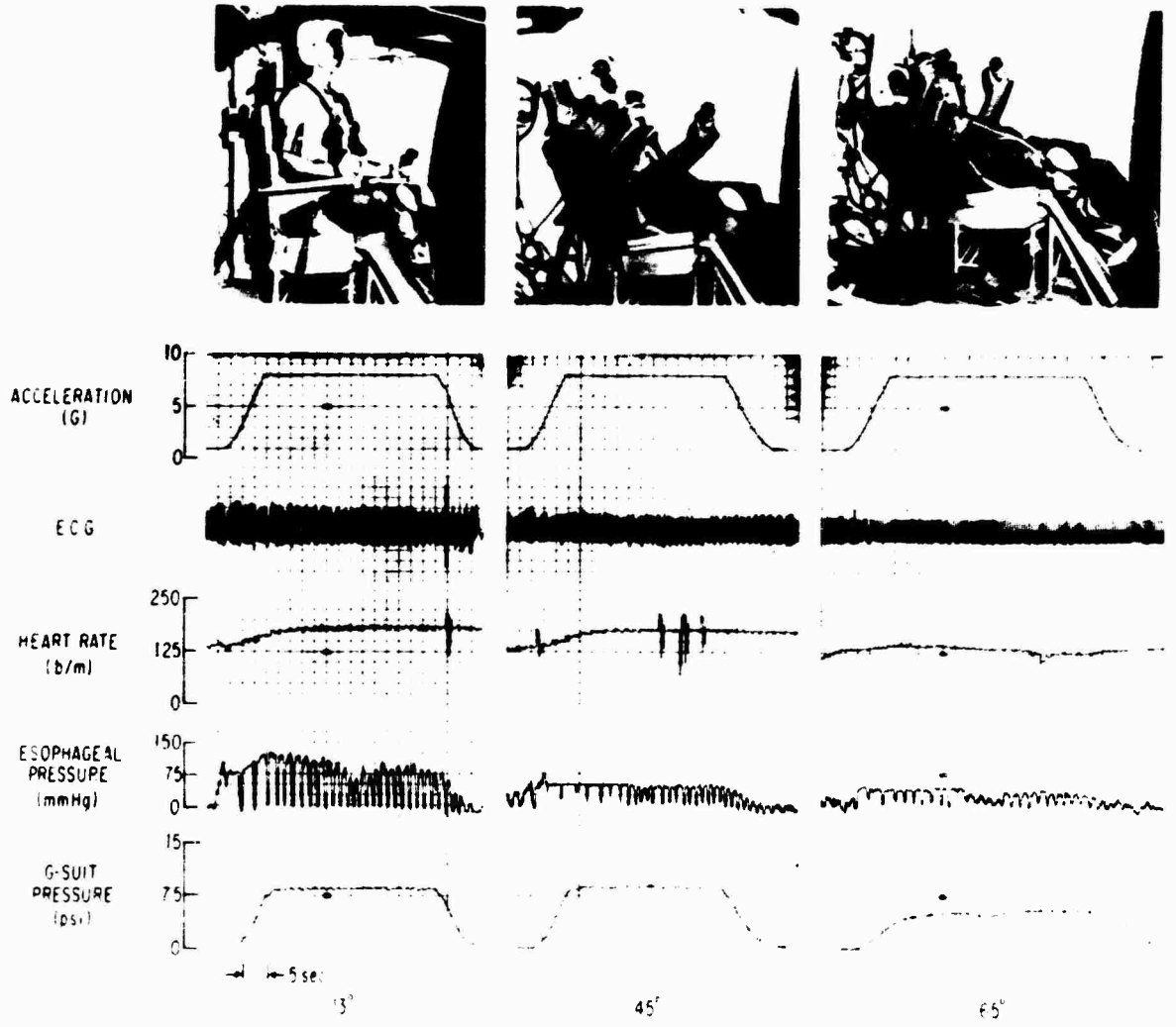


Figure 26. Physiologic responses to tilting backward at high sustained G. (From Burns, 1975)

Subject performance is inversely related to level of G exposure and rapidly deteriorates above +6 G_z. Using target tracking tasks the efficacy of the tilt-back seat in increasing performance has been evaluated. The seat angle was varied between 30° and 65° and the subject's performance was scored as percent target hit score at various G-levels and seat angles. Significant improvements in performance were noted at 45° and 65° when compared to a more upright 30° seat angle (Figure 27).

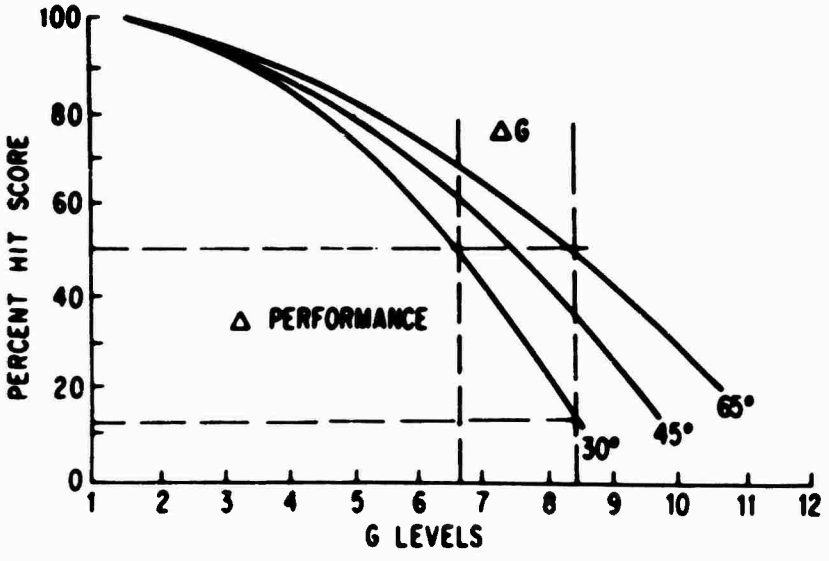


Figure 27. Improved target-tracking performance obtained by tilting backward. (From Rogers et al., 1973)

Pathophysiology of HSG

Swine have been shown to be an excellent human analog for acceleration research: (1) they have the same heart-to-eye distance as man; (2) they spontaneously perform an M-1 straining maneuver with arterial blood pressure responses similar to those in man (Figure 28); and (3) they have acceleration tolerances similar to man's. Six swine wearing inflated anti-G suits have been exposed to +5, 7, and 9 G_z for 45 seconds and euthanized and autopsied immediately after the runs. Subendocardial hemorrhage was found in all animals; however, results from animal experimentation are difficult to extrapolate to man.

Myocardial enzyme studies and serial vectorcardiograms have been conducted on human volunteer subjects exposed to a maximum of +9 G_z for 45 seconds. No evidence of myocardial damage has been found.

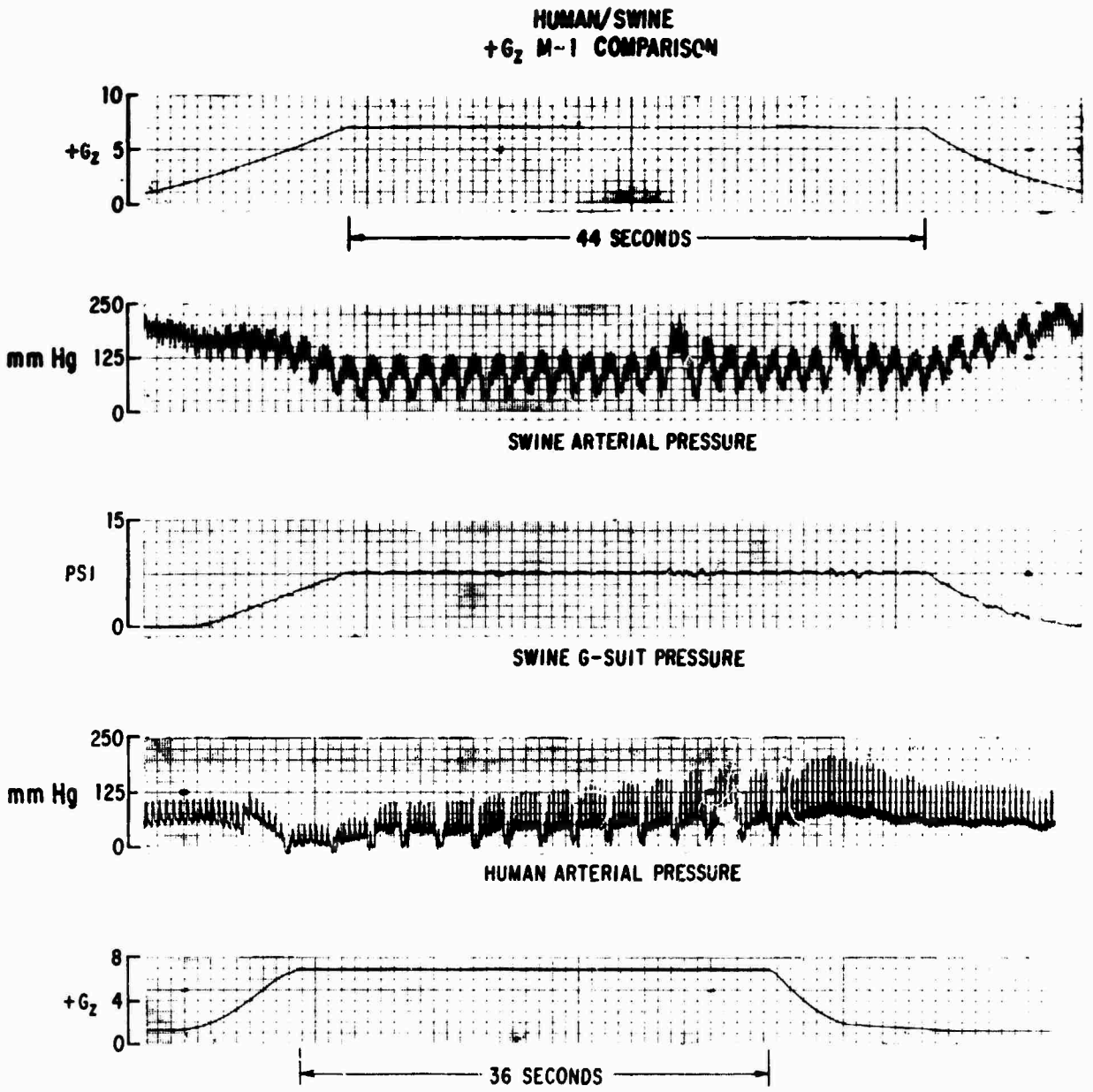


Figure 28. Human/swine $+G_z$ M-1 comparison.

EFFECTS OF $-G_z$

$-G_z$ (inertial force directed from foot toward the head) will result in an increased arterial pressure at head level. The pressure within the veins outside the cranial cavity becomes precipitously high, and may be sufficient to rupture the thin-walled venules. Intracranial venous pressure rises, but is counterbalanced by a concomitant rise in intracranial cerebrospinal fluid pressure, so there is little actual danger of intracranial hemorrhage. Hemorrhages within the eye present the primary source of damage from $-G_z$. $-G_z$ causes distention of the jugular veins and veins of the sinuses and conjunctivae. A sharp rise occurs in both arterial and venous pressures at head level and leads to transudation of fluid from the blood into the tissue spaces of the head and neck. Also, return of blood to the heart becomes inadequate because of loss of effective blood volume.

As the pressure in the vessels of the neck increases during $-G_z$, the carotid sinus reflex and reflexes initiated by low-pressure-system volume receptors cause a slowing of the heart and a dilation of the arterioles. The reduced heart rate and decreased total peripheral resistance (TPR) cause the arterial pressure to approach the venous pressure. As the pressure gradient across the capillaries declines, cerebral blood flow decreases. This may result in cerebral stagnant hypoxia if the acceleration is prolonged.

Sudden acceleration producing a force of $-3\ G_z$ is considered the upper limit of human tolerance, although human experiments at $-G_z$ are very few. When such a force is applied, venous pressures of the order of 100 mm Hg develop, leading to small conjunctival bleeding areas and marked discomfort in the head and neck. On the other hand, the pressure-protected cerebral vessels, enclosed in the skull and bathed in cerebrospinal fluid, show no deviation from their normal caliber. Thus, there is no danger of cerebrovascular damage as long as the skull remains intact.

During $-G_z$ maneuvers it has been reported that vision may redout. Although no cases of this condition have occurred during experimental work, it is possible that, in aircraft, vision may be obscured by the gravitation of the lower eyelid over the cornea. The muscles in the lower eyelid are relatively weak due to the tendency of gravity to normally hold it down. The covering of the eyes by a red curtain (since the vessels of the eyelid are engorged) may be responsible for the reports of redout during $-G_z$.

Actually, $-G_z$ does not present much of a problem in military flying because aircraft are not stressed for high $-G_z$ loads and hence pilots avoid it in their maneuvers whenever possible.

EFFECTS OF $\pm G_x$

The gravitoinertial forces acting at right angles to the long axis of the body in a transverse (anterior-posterior or posterior-anterior) direction are annotated by the symbol $\pm G_x$. The prone and supine positions of the body provide the greatest tolerance to acceleration because of the limited hydrostatic effect on blood pressure--note section on the tilt-back seat earlier in this review ($+G_z$).

Centrifuge studies have shown that the effects of $\pm G_x$ on the cardiovascular system are much less than those of $\pm G_z$. During transverse acceleration, subjects have withstood up to +15 G_x without blackout, indicating effective blood pressure to the head and eyes. However, performance capabilities are impaired at levels as low as +5 G_x, where it becomes impossible to raise the head and extremely difficult to raise the arms against the high G force. Some limb movement can be achieved between +5 and +9 G_x, but it is always influenced by the G field. For example, it may be impossible for a pilot to reach forward and activate a switch located on the instrument panel, whereas fine control using wrist and finger movement is possible and can be accomplished if the arm is not required to be moved away from the armrest (advanced fighter-type aircraft will utilize this armrest-controller "fly-by-wire" capability).

Visual disturbances such as blurring and excess lacrimation have been noted above +12 G_x. The decrease in vision above this level may in part be attributed to eyeball distortion caused by the large force acting on the forward portion of the eye.

Above +8 G_x, respiration becomes increasingly difficult because the rib cage becomes more and more fixed in the expiratory position and diaphragmatic breathing is required to maintain sufficient air exchange. Also, $\pm G_x$ induces ventilation/perfusion inequalities similar to those observed during $+G_z$. Some subjects have been able to withstand transverse acceleration of +26 G_x for several seconds with complete post-run recovery, and 9 relaxed subjects have tolerated +10 G_x for a mean duration of 113 sec. Painful respiration is usually the limiting factor in human tolerance

to $\pm G_x$, and time-tolerance at various levels of acceleration is usually defined by this difficulty in breathing. Some of these respiratory difficulties at $+G_x$ may be eased by positive pressure breathing, which extends the tolerable time at +10 G_x by approximately 70%.

PERCEPTION OF MOTION AND POSITION

Spatial orientation and postural equilibrium are maintained by the combined activities of the visual, vestibular, kinesthetic, tactile, and auditory systems as they monitor intensities and directions of various physical energies acting on the body. By far the most important of these senses from the standpoint of precision, reliability, and overall utility is the visual system. The vestibular system, which developed to insure the stability of the visual system and the body in general, is used in the absence of vision as a backup equilibratory system. A similar subordinate function is served by the kinesthetic, tactile, and auditory systems, although with considerably less precision and reliability. Whereas the basic principles of the functioning of the visual system are clearly appreciated by most people, the principles of vestibular function are not; for that reason, a brief discussion of vestibular function is provided in the following sections to give the reader the necessary background for understanding the mechanisms of spatial disorientation in flight.

Vestibular Anatomy

Within the petrous portion of each temporal bone is a tortuous excavation known as the bony labyrinth, which is filled with perilymph, a fluid much like cerebrospinal fluid in composition. The bony labyrinth consists of three main parts: the cochlea, the vestibule, and the semicircular canals. Within each part of the bony labyrinth is a part of the delicate, tubular, membranous labyrinth, which contains endolymph, a fluid characterized by its relatively high concentration of potassium. In the cochlea the membranous labyrinth is called the cochlear duct or scala media, and this organ converts acoustic energy into neural information. In the vestibule lie the two otolith organs, the utricle and the saccule. They translate gravitational and inertial forces into spatial orientation information--specifically, information about linear motion and position of the head. The semicircular ducts, contained in the semicircular canals, convert inertial torques into information about angular

motion of the head. Figure 29 illustrates the gross anatomy of the inner ear. Observe that the three semicircular canals and their included semicircular ducts are oriented in three mutually perpendicular planes. This allows the most efficient analysis of angular motion in any direction, and gives rise to the names of the canals: anterior vertical (or superior), posterior vertical (or posterior), and horizontal (or lateral).

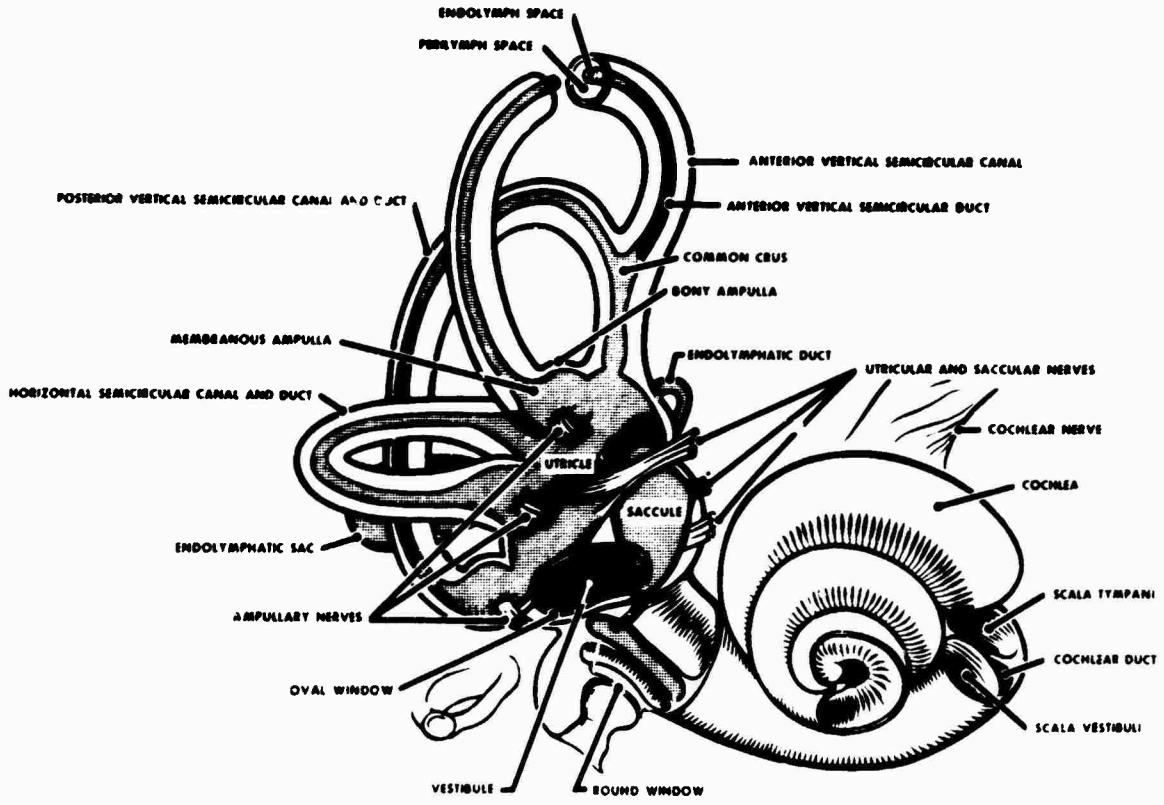


Figure 29. Anatomy of the inner ear.

The semicircular ducts communicate at both ends with the utricle, and one end of each duct is dilated to form an ampulla. Inside each ampulla lies a crest of neuroepithelium, the crista ampullaris; atop the crista, occluding the duct, is a gelatinous structure called the cupula (Figure 30). The hair cells of which the crista ampullaris is composed project their cilia into the base of the cupula, so that whenever the cupula moves, the cilia are bent.

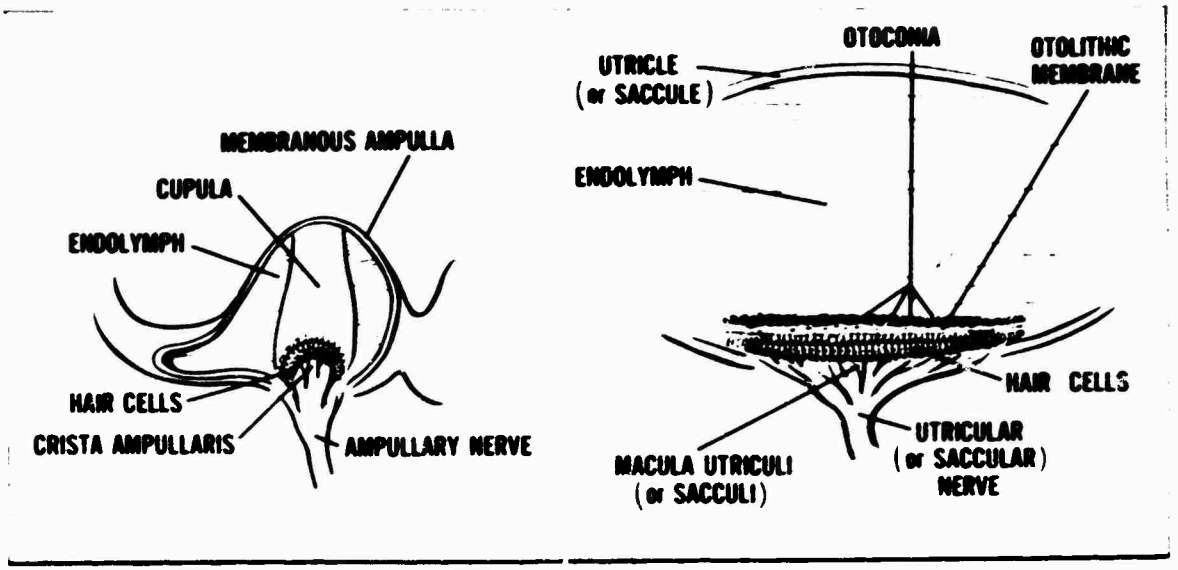


Figure 30. Microanatomy of the vestibular end-organs.

Lining a part of the bottom of the utricle is another patch of neuroepithelium, the macula utriculi, and on the medial wall of the saccule is found still another, the macula sacculi. The cilia of the hair cells comprising these structures project into overlying otolithic membranes, one for each macula (Figure 30). The otolithic membranes consist of many tiny calcium carbonate crystals, called otoconia, suspended in a gelatinous matrix. The otolithic membranes, having almost three times the density of the surrounding endolymph, displace endolymph and shift position relative to their respective maculae when subjected to changing gravitoinertial forces; this shifting of relative position results in bending of the cilia of the hair cells of the maculae. The cupulae, having no otoconia and being of approximately the same density as endolymph, cannot respond to such forces, but respond instead to the inertial torques of the rings of endolymph contained in the semicircular ducts.

The utricular nerve, the saccular nerve, and the three ampullary nerves converge to form the vestibular nerve, a portion of the VIIIth cranial or statoacoustic nerve (Figure 31). The vestibular nerve projects

to the four vestibular nuclei (superior, medial, lateral, and inferior) in the brain stem, and to the cerebellar cortex, where correlations and computations utilizing vestibular, visual, and other sensory data are made. The information emerging from the vestibulocerebellar axis, as the vestibular end-organs and their immediate central projections are collectively called, is fed to the motor nuclei of the extrinsic eye muscles (oculomotor, trochlear, and abducens) via the medial longitudinal fasciculus, and to the motor nuclei of the neck and other skeletal muscles via the medial longitudinal fasciculus and the lateral vestibulospinal tract. These two important efferent neural pathways mediate the vestibulo-ocular and postural reflexes. Other efferent connections of the vestibulocerebellar axis include projections into the brainstem reticular formation and to the vestibular projection areas of the cerebral cortex.

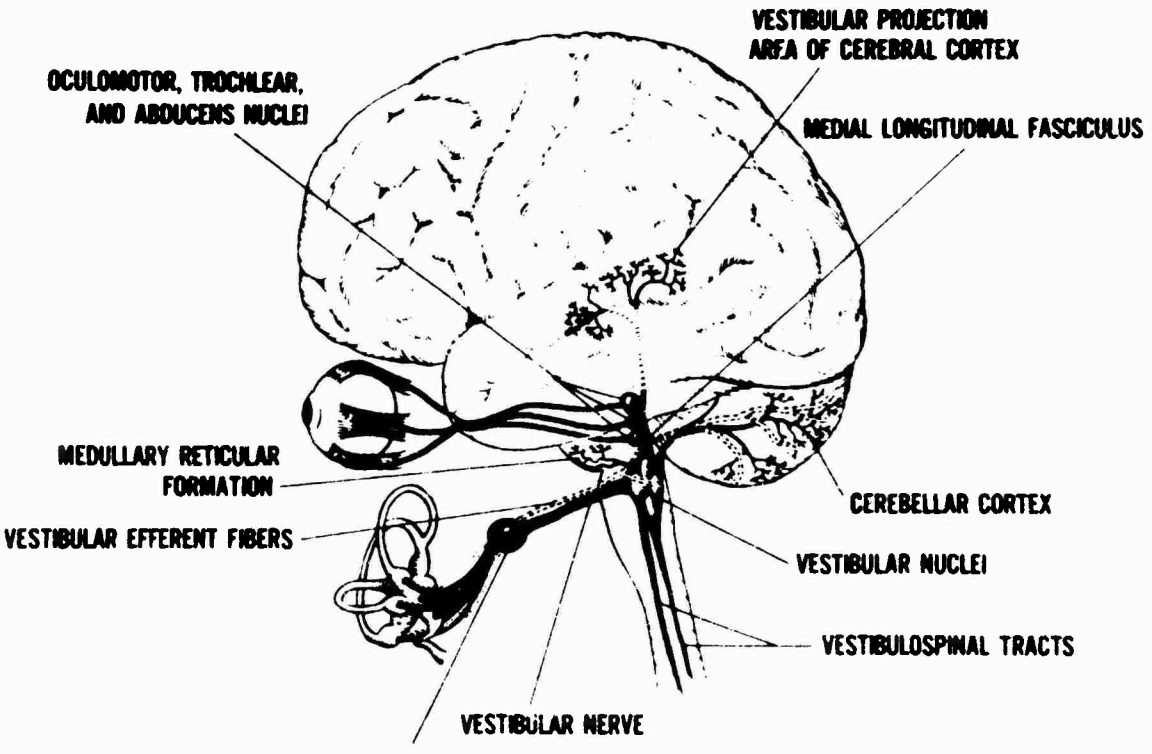


Figure 31. Neural connections of the vestibular system.

A rather fascinating projection, the vestibular efferent system, carries neural information from the vestibular nuclei and cerebellum out to the hair cells of the cristae and maculae, undoubtedly to exert some form of control over afferent vestibular signals.

Vestibular Mechanics

The hair cell is the operational unit, so to speak, of the vestibular system: it converts spatial and temporal patterns of mechanical energy applied to the head into neural information. How this is done is shown in Figure 32. Notice that each hair cell possesses one relatively large kinocilium on one side of the top of the cell and up to a hundred smaller stereocilia on the same surface. The hair cell exhibits, therefore, morphologic polarization; i.e., it can be oriented in a particular direction. When the cilia are deviated in the direction of the kinocilium, the hair cell depolarizes, and the frequency of action potentials generated in the vestibular neuron attached to the hair cell increases above a certain resting frequency--the greater the deviation of the cilia, the higher the frequency. When the cilia are bent away from the side of the kinocilium, the hair cell hyperpolarizes, and the frequency of action potentials in the corresponding neuron in the vestibular nerve decreases. The same basic process occurs in all the hair cells in the three cristae and both maculae; the differences lie in the physical events that cause the deviation of cilia and in the directions the various groups of hair cells are oriented.

The otolith organs respond to linear accelerations and gravity; the semicircular ducts respond to angular accelerations. This statement, pictorialized in Figure 33, is the cardinal principle of vestibular mechanics.

The otolith organs monitor the magnitude and direction of the gravitoinertial force resulting from linear acceleration and gravity by registering the magnitude and direction of movement of the otolithic membranes, as they assume their positions over their respective maculae in response to the gravitoinertial force. Figure 34 shows as an example how a portion of the utricle responds to a changed direction of the force of gravity, resulting from a backward tilt of the head, by transmitting an increased frequency of action potentials from that segment of macular neuroepithelium. Forward tilting, of course, has the opposite effect. One can also appreciate from Figure 34 how purely inertial forces resulting from linear acceleration can stimulate the utricular and saccular maculae. The combined activity of all portions of all the maculae provides the central nervous system with information about gravitoinertial forces acting in virtually any direction.

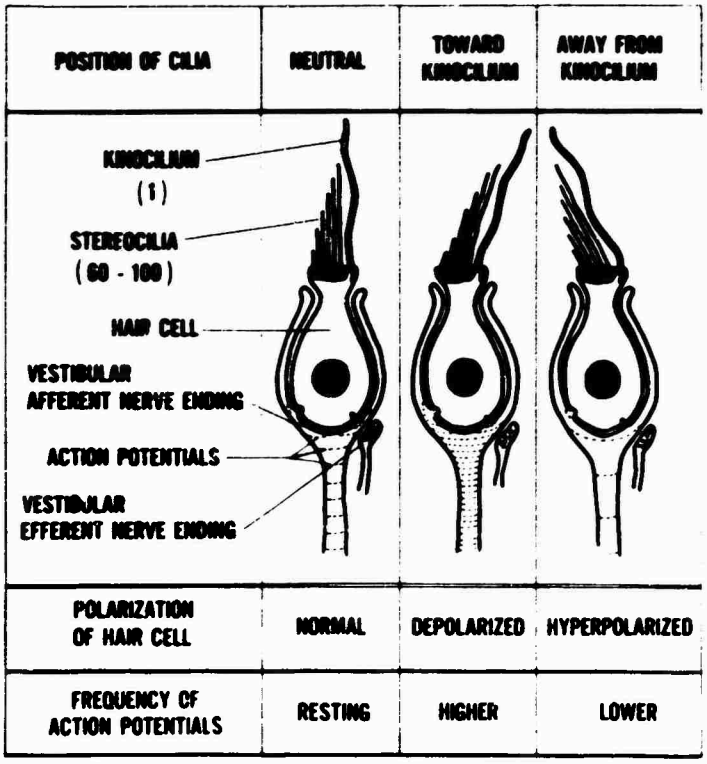


Figure 32. Function of a vestibular hair cell.

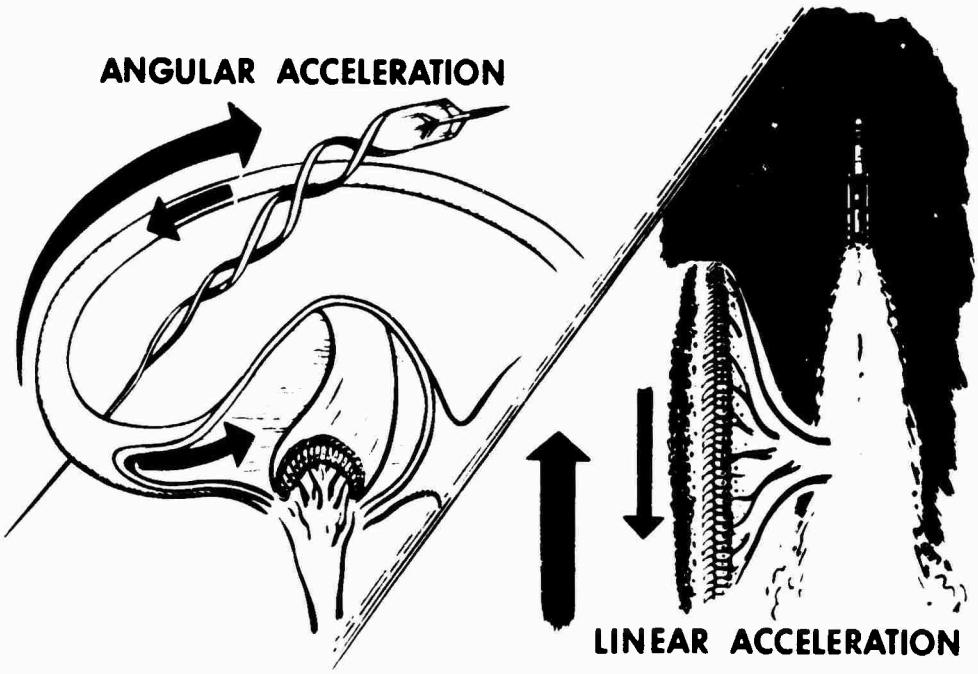


Figure 33. The otolith organs respond to linear accelerations and gravity; the semicircular ducts respond to angular accelerations.

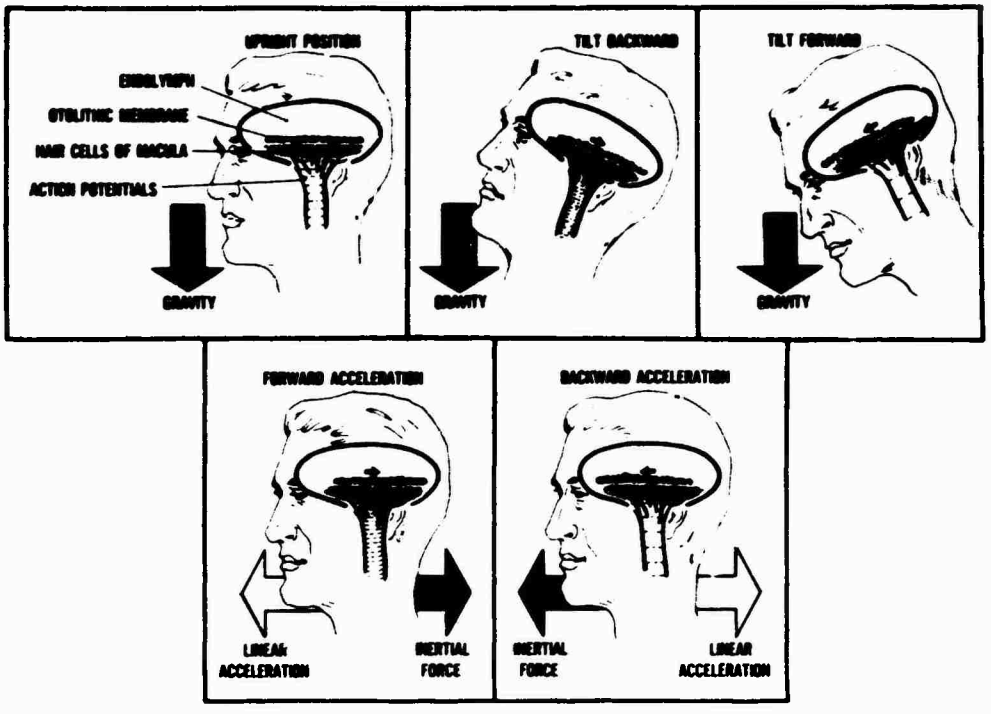


Figure 34. Mechanism of action of an otolith organ.

The action of the cupula and crista of the right horizontal semicircular duct, as it responds to a brief, clockwise turn of the head, is illustrated in Figure 35. The inertial torque of the endolymph, resulting from the angular acceleration of the head, deviates the cupula in the direction of the kinocilia of the hair cells of the crista, thus increasing the frequency of action potentials in the corresponding ampullary nerve. When the head decelerates to a stop, the cupula is driven back to its resting position by the inertial torque resulting from the angular momentum gained by the endolymph during the brief acceleration. The same sequence of events occurs in the opposite (left) horizontal semicircular duct, except that the cupula bends the cilia away from the kinociliary poles of the hair cells, thereby causing a reduction in the frequency of action potentials in the left horizontal ampullary nerve. The other two pairs of semicircular ducts monitor angular accelerations in a similar "push-pull" fashion, so that rotations of the head in any spatial plane can be monitored by at least one pair of semicircular ducts.

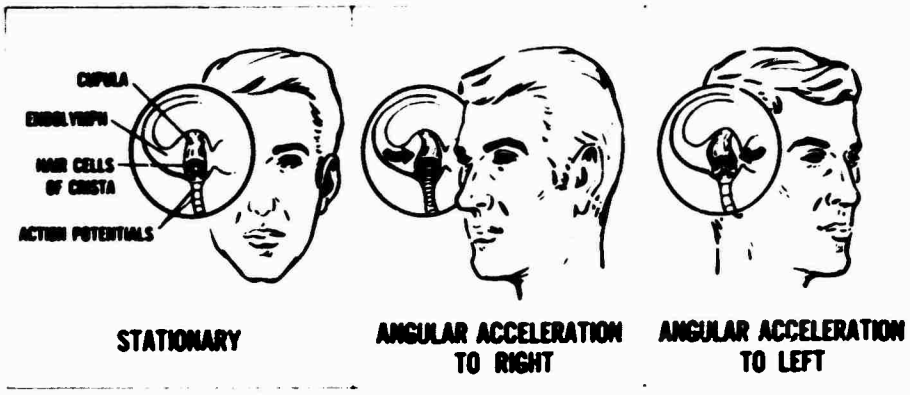


Figure 35. Mechanism of action of a semicircular duct.

It should be pointed out that the otolith organs cannot respond to constant linear velocities, nor can the semicircular ducts respond to constant angular velocities. An example portraying the latter deficiency will be helpful (Figure 36). Because the cupula deviates when an angular acceleration is imparted to the head, one acquires a percept of angular motion, the of turning in the direction opposite the cupular deviation. When the angular acceleration ceases, leaving the body turning at a constant angular velocity, the inertial torque of the endolymph is removed from the cupula, and the cupula returns over a period of several seconds to the neutral position because of elasticity in the system. The cupula having returned, the sensation of turning disappears, despite the fact that turning continues. If one then decelerates to a stop, the angular momentum of the moving endolymph causes a pressure to be exerted on the cupula in the opposite direction from that in which the cupula deviated during acceleration; the resulting cupular deflection now signals a turn in the opposite direction from the actual angular motion of the body. After the stop the cupula again returns to a neutral position and the sensation of turning disappears.

The belaboring of the above example was to illustrate the fact that the vestibular system is capable of making errors. The system provides accurate spatial orientation information when stimulated by the patterns of acceleration encountered in our usual ground-based activities like walking, running, jumping, and making quick head movements; but when abnormal patterns of acceleration are encountered, as in flight, the

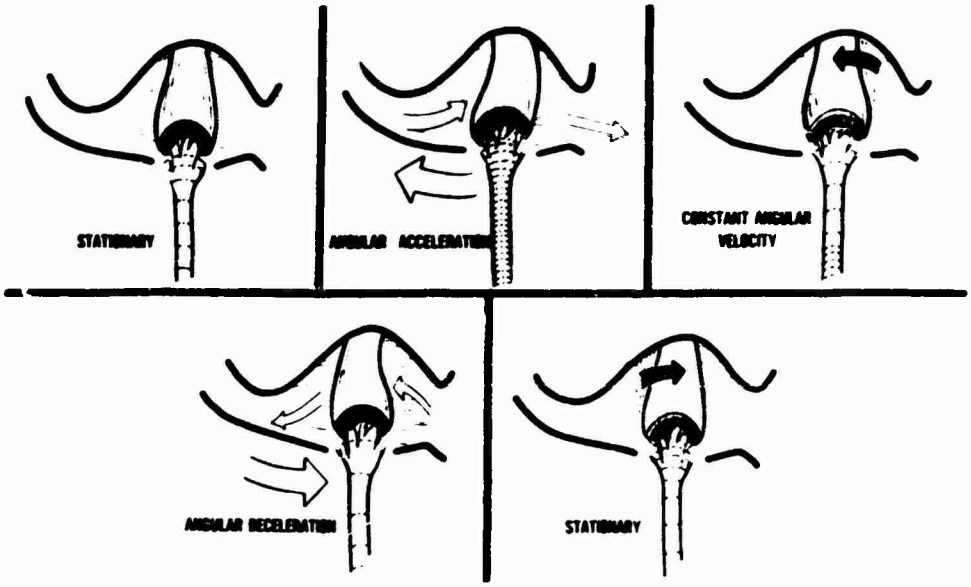


Figure 36. Effect of constant angular velocity on the semicircular-duct system--erroneous velocity information is generated.

system generates erroneous information. In general, those patterns of acceleration containing low-frequency components result in inaccurate percepts of motion and position: the frequency response of the vestibular apparatus is such that perceptual distortion results when the system is used to monitor acceleratory signals below the designed frequency bandwidth of the system.

Vestibular Reflexes

The vestibulo-ocular reflexes help to stabilize the retinal image. The reflexes originating in the semicircular ducts result in movements of the eyes in the opposite direction from angular movements of the head; i.e., the gaze "locks onto" the inertial environment, as the eyes are driven with an angular velocity in the direction of, and proportional to, cupular displacement. Such eye movements are called compensatory; they are interspersed with rapid anticompensatory eye movements in the direction of rotation of the head, which serve to bring the eyes quickly to a new position of gaze. When the compensatory and anticompensatory eye

movements continue for several beats, the resulting oscillating movement of the eyes is called nystagmus, with the compensatory and anticompensatory movements being called the slow and fast phases of the nystagmus, respectively (Figure 37). Instead of being a help, nystagmus is often a hindrance in flight, since the relatively prolonged (i.e., low frequency) angular accelerations required to produce nystagmus can render the vestibular system incapable of providing accurate information for tracking the external world, and any reflex movement of the eyes makes visualizing the cockpit interior somewhat difficult. In extreme situations, such as a spin, nystagmus can make both the cockpit and the external world appear as a blur, thus compromising effective recovery.

The vestibulo-ocular reflexes of otolith-organ origin also appear to aid in stabilization of the retinal image. The elevator reflex results in a downward shift of gaze in response to an upward linear acceleration, and vice versa. A similar reflex seems to be operative in response to

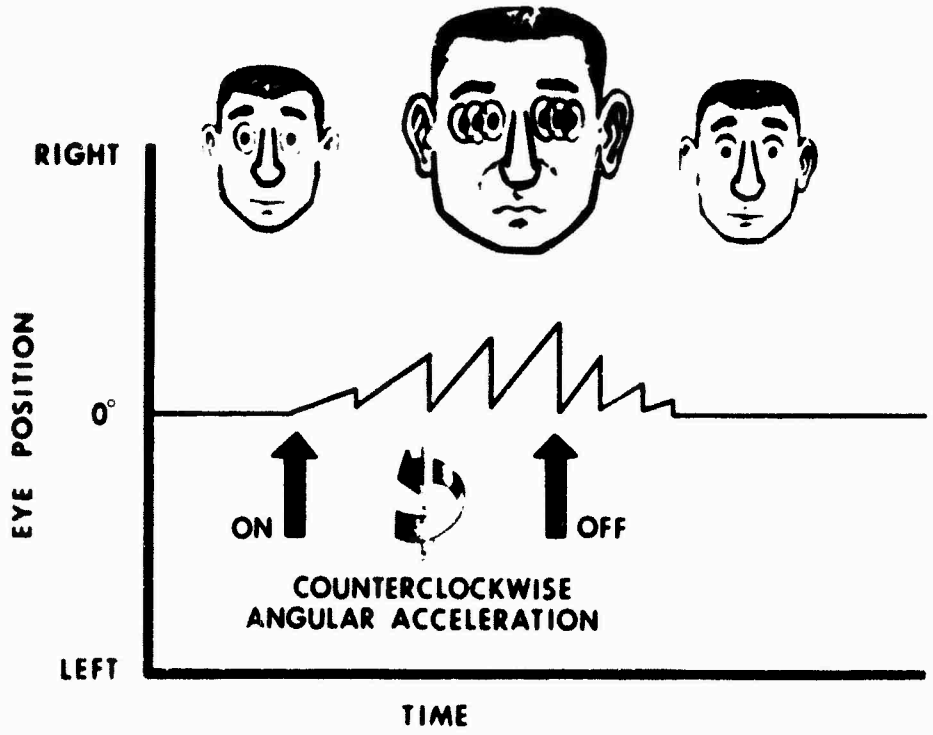


Figure 37. Vestibular nystagmus.

sideward linear accelerations. When the head is tilted in the y-z (frontal) plane, the ocular counterrolling reflex generates a sustained angular displacement of the eyes about the visual (anteroposterior) axis in the opposite direction of the tilt, but with by no means compensatory amplitude.

Vestibulospinal or postural reflexes are involuntary movements of the head, limbs, and trunk that are directed toward maintaining the head as a stable platform for the eyes and protecting the body from injury during a fall. They are generated by linear and angular accelerations; but, in comparison to vestibulo-ocular reflexes they can more readily be overridden by voluntary actions.

Vestibulovegetative reactions is a name given to a poorly understood group of reflexes involving autonomic activity generated by vestibular stimulation. It has been suggested that such autonomic reflexes serve to prepare the cardiovascular system for the inertial effects of acceleration, and that motion sickness results when these reflexes are evoked in excess by abnormal amounts of vestibular stimulation; this theory has not been proved, however.

Habituation and Visual Dominance

One might infer from the foregoing discussion of vestibular function that a given acceleratory stimulus always results in a given motor or perceptual response. Such is not the case. If one undergoes, for example, repeated angular accelerations of a particular type over a period of hours to days, a definite decrement in nystagmic response, called habituation, is obtained. Changing a person's state of mental arousal has also been shown to affect profoundly the quantity and quality of his vestibulo-ocular reflexes--exaggerated reflexes being associated with the hyperaroused state, and even absent reflexes being associated with the completely relaxed state in some individuals. Of great importance from the standpoint of flying safety are the concepts of visual dominance, a state wherein one is able to gain virtually all of his orientation information through his visual system, and vestibular suppression, a process by which vestibulo-ocular reflexes are reduced or abolished while one orients visually. Both visual dominance and vestibular suppression are developed by working in an environment of conflicting visual and vestibular cues, as in instrument or formation flying, and they are maintained by repeated exposure to such environments.

SPATIAL DISORIENTATION

A pilot who does not correctly perceive his position, attitude, or motion relative to the earth is said to have spatial disorientation if a correct percept is required for proper control of his vehicle; a person who has a false impression about his position, attitude, or motion, but an accurate percept is irrelevant to his task, is simply unoriented. The term, pilot vertigo, is used synonymously with spatial disorientation, even though the conventional medical use of the word "vertigo" implies a strong but false sensation of angular or linear motion. A distinction is sometimes made between unrecognized spatial disorientation, in which the pilot is unaware of his erroneous impression, and recognized spatial disorientation, in which he is aware of the fact that he is disoriented but is unable to dispel his false percept. Of the two conditions, the former is to be feared more than the latter, as one can still control his aircraft, or eject, if he knows he has vertigo.

The importance of spatial disorientation as a cause of aircraft accidents is periodically substantiated by studies of USAF accident statistics. Nuttall and Sanford (1956) reported that, in USAFE during the period 1954-1956, spatial disorientation accidents accounted for 4% of major accidents and 14% of fatal accidents. Moser in 1969 reported a study of accidents in ADC during the four-year period, 1964-1967: he found spatial disorientation to be a significant factor in 9% of major accidents and 26% of fatal accidents. Barnum and Bonner (1971) reviewed all the spatial disorientation accidents in the USAF from 1958 through 1968 and found that in 281 or 6% of the 4,679 major accidents, spatial disorientation was a causative factor: fatalities occurred in 211 of the spatial disorientation accidents, accounting for 15% of the 1,462 fatal accidents. Barnum repeated his study for the three-year period, 1969-1971: he concluded (1973) that spatial disorientation accidents again accounted for 6% of total major accidents, but for only 10% of fatal accidents. Kellogg, in an independent 1973 study, found the annual incidence of spatial disorientation accidents in the years 1968 through 1972 to range from 4.8% to 6.2%, and confirmed the relatively high proportion of fatalities in accidents resulting from spatial disorientation. A comment made by Barnum and Bonner in their first study summarizes some interesting data about the "average pilot" involved in a spatial disorientation accident: "He will be around 30 years of age, have 10 years in the cockpit, and have 1500 hours of first-pilot/instructor-pilot time. He will be a fighter pilot and will have flown approximately 25 times in the three months prior to his accident."

Causes of Spatial Disorientation

When flying primarily by the use of an external visual reference, such as the earth below, maintaining spatial orientation is usually not a difficult task. At night or in instrument meteorologic conditions (IMC, otherwise known as "weather"), one uses his aircraft instruments to orient himself. Chief among those instruments is the attitude indicator, which provides the pilot with a moving pictorial representation of his aircraft in relation to the distant horizon (Figure 38). Other instruments give indications of engine power, angle of attack, airspeed, altitude, vertical velocity, rate of turn, heading, geographic position, and other parameters. The key to success in instrument flying is the development of an efficient instrument cross-check, the process in which the pilot alternately samples information from control instruments, performance instruments, and navigation instruments, making corrections on the control instruments to minimize errors in the performance and navigation instrument indications. (See Chapter 7 of AF Manual 51-37, Instrument Flying, for detailed information on proper instrument cross-check technique.) As might well be imagined, if a pilot is unable to see, interpret, believe, and process the information presented by the flight instruments during times when those instruments are the pilot's sole source of accurate orientation information he will become disoriented.

Formation flying presents special problems to the pilot with regard to maintaining spatial orientation. While flying in formation the pilot must allow the lead aircraft to be his only attitude reference; he must trust and follow the leader, watching him and making whatever control movements are necessary to maintain the proper position with respect to the lead aircraft. Formation flying in weather or at night can completely deprive a pilot of visual reference to the earth, because the lead aircraft does not function as an indicator of the horizon during turns, climbs, and dives, and the pilot has little time to be scanning his own instruments while trying to stay "locked on" the aircraft ahead of him. Small wonder, then, that so large a proportion of spatial disorientation accidents occur during night and weather formation flights.

The classic approach to a discussion of the mechanisms responsible for spatial disorientation is to categorize certain illusions that have been described in flight according to whether they arise primarily from stimulation of the vestibular, the visual, or some other sensory system. The illusions considered vestibular in origin are further divided into those attributable to inadequacies of the semicircular-duct system and those resulting from inadequacies of the otolith-organ system.

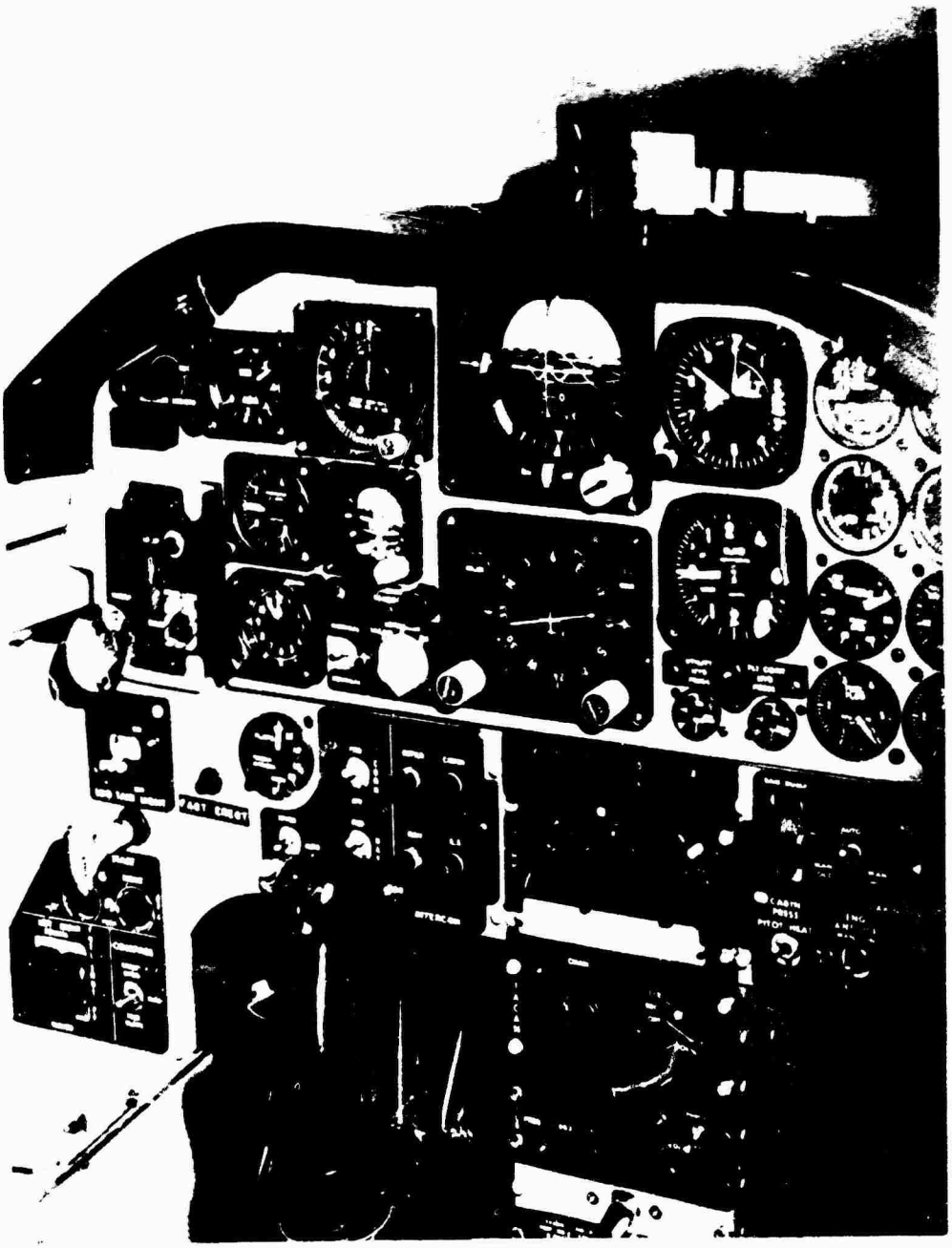


Figure 38. The instrument panel of a T-38. The attitude indicator is at the top center of the panel.

The most commonly reported form of spatial disorientation is called the leans. The pilot suffering from this condition feels himself angularly displaced from his actual (usually upright) position in the roll plane, and develops a strong urge to lean in the direction of his falsely perceived vertical in an attempt to assume an upright posture--whence the name. At least three explanations for this phenomenon are forwarded. One suggests that the leans is created when the pilot undergoes an unperceived (subthreshold) roll in one direction and then recovers from the unwanted resulting bank by means of a perceived (suprathreshold) correcting roll, with his net percept being that of a roll into a bank in the direction of the suprathreshold roll. For example, if a pilot allowed his aircraft to roll to the left at a rate of about 2°/sec for 10 seconds while he was occupied with some distracting task, then recognized the unwanted 20° bank to the left and recovered immediately with a 20°/sec roll to the right for one second, the net percept he would have would be one of having made a roll to the right; he would, we hope, fly the aircraft properly by the instruments, but would likely lean to the left to help alleviate the feeling of being in right bank (Figure 39). It has also been suggested that the leans can result from a varying roll-perception threshold. Even though it has been stated that, for perception of angular motion to occur, the product of angular acceleration and time of application of that acceleration must be equal to or greater than a constant (viz., Mulder's constant--2.5°/sec), we recognize that the threshold actually varies considerably, depending not only on the plane of the angular motion but also on the state of arousal of the pilot. Should a complacent, daydreaming pilot have an elevated threshold for perception of angular motion, he may miss a 4°/sec roll to the left; but, jarred from his quiescent mental state by the recognition of the unusual attitude resulting from the unperceived roll, he would perceive the returning roll to the right quite easily, even though it may be below his usual roll-perception threshold. The third possible mechanism involved in the production of the leans is that of resetting one's perception of the vertical during a prolonged constant-rate banked turn, such as a turn in a holding pattern. During the constant-rate turn, the sensation of turning disappears as the cupula-endolymph system returns to the resting position; meanwhile, the gravitoinertial force vector conveys only that "down" is toward the floor of the aircraft. Under such circumstances the pilot may lose his impression of being in a bank, and may experience the sensation of rolling into a bank in the opposite direction when he finally rolls out of the banked turn he was in.

A second type of vestibular illusion resulting from stimulation of the semicircular-duct system is the somatogyral illusion. The subject with

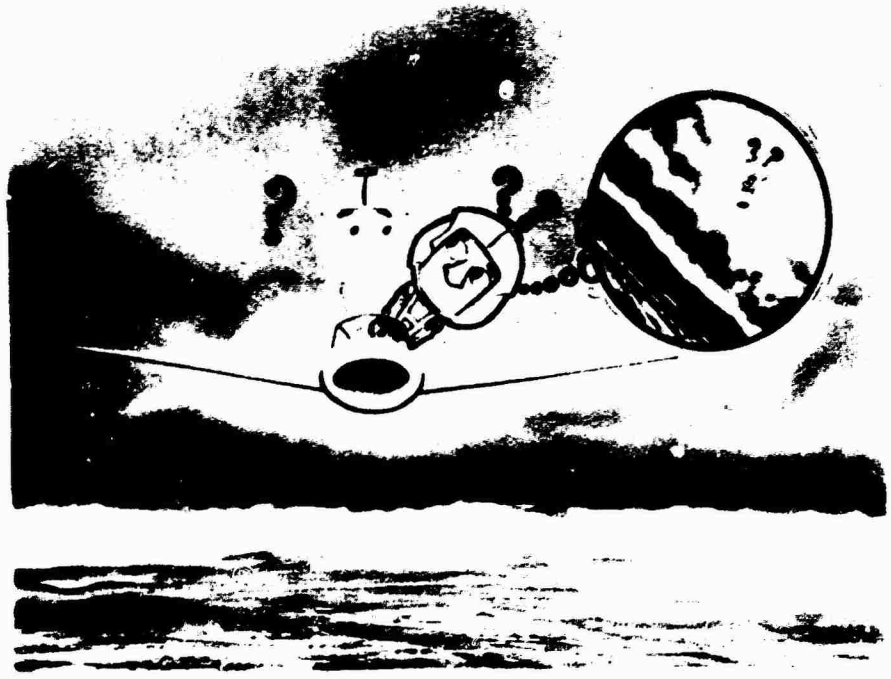


Figure 39. The leans.

this illusion has a sensation of rotation in a direction or of a degree different from that of the actual motion occurring. (The term oculogyral illusion, which has on occasion been used in a more general sense than originally intended, refers to the apparent movement of a viewed object, such as an instrument panel, occurring in response to stimulation of the semicircular-duct system.) The graveyard spin (Figure 40) is an example of the effect of the somatogyral illusion in flight. A pilot who has gotten into a spin in weather falls victim to the graveyard spin if the following sequence of events occurs: (1) he allows the spin to continue long enough for his semicircular-duct system to partially or completely return to the neutral condition, which results in a decreased or absent sensation of spinning; (2) he initiates corrective action by pushing opposite rudder to stop the spin, whereupon he falsely perceives a spin in the direction opposite that of his original spin; so (3) he re-enters the original spin, because he feels no spin during the time he is spinning, but feels as though he is going into a spin whenever he tries to stop the existing spin. Much more likely to occur than the graveyard spin is the graveyard spiral, which also owes its existence to the somatogyral illusion.

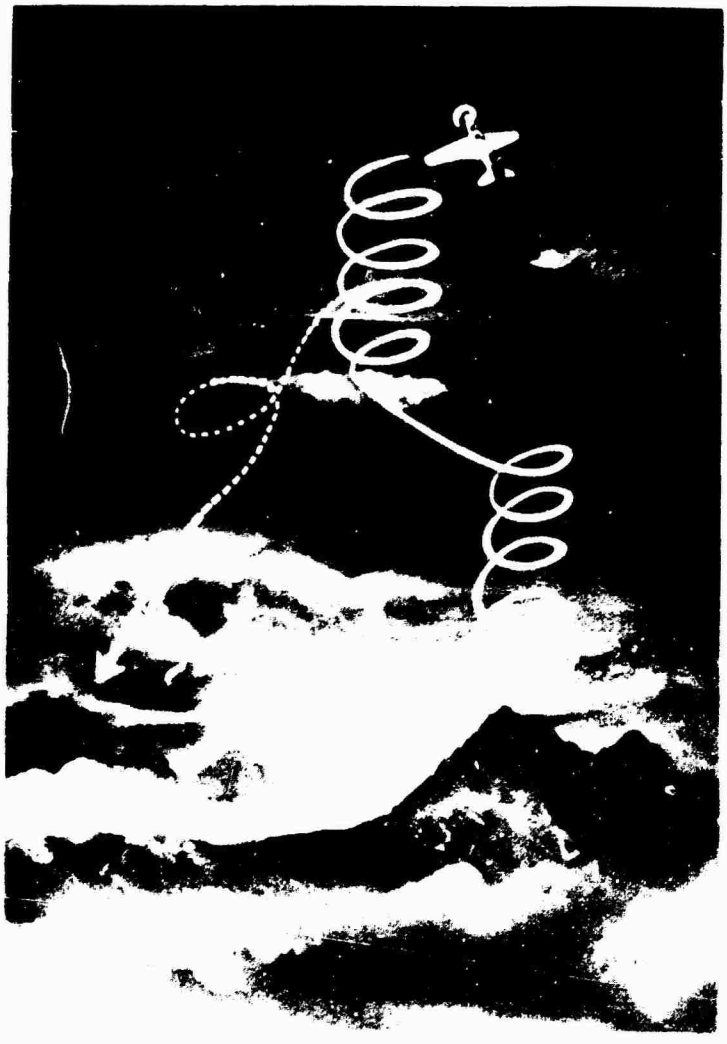


Figure 40. One form of the somatogyral illusion--the graveyard spin. The dotted line indicates perceived motion; the solid line indicates actual motion.

In the graveyard spiral the aircraft is flying, not spinning, but is in a steeply banked attitude in a downward spiral of increasing tightness. The graveyard spiral is entered when a pilot is unable to resolve correctly a conflict between his false perception of being upright and an instrument indication that he is in a fairly steep bank, or when the instrument indication of the bank is ignored altogether. In the banked attitude the

aircraft maintains a more or less constant rate of turn but loses altitude; so the pilot, not perceiving the turn because his semicircular ducts cannot respond to constant angular velocities, adds power and pulls back on the control stick to try to stop the descent. This maneuver, however, serves to tighten the spiral and add to the G force acting on the pilot and the aircraft. Unless the pilot levels the wings, the situation can only deteriorate further; but if he does level the wings and stop the angular motion of the spiral, he feels as though he is entering a steep bank and rapid turn in the opposite direction. This unacceptable feeling of banking and turning during the attempted recovery must be recognized as an illusion, or the pilot will be unable to extricate himself from his downward spiral.

The vestibular Coriolis effect (Figure 41) and the resulting Coriolis illusion occur when one moves his head in a plane that cuts across the plane of rotation of an already rotating system. Consider, for example, a person who has been rotating at a constant angular velocity in the yaw plane long enough for the endolymph to "come up to speed" in his horizontal semicircular ducts: the cupulae in the ampullae of those ducts have returned to their resting positions and the sensation of horizontal angular motion has disappeared. If he then nods his head downward in the pitch plane, he is taking his horizontal canals out of the plane of rotation and is putting his two sets of vertical canals at least partially into the plane of rotation. The rotational inertia of the endolymph in the horizontal semicircular ducts has to be dissipated, however; and in the process, the cupulae in the horizontal ducts are deviated, resulting in a sensation of angular motion in the new plane of the horizontal canals, which is now the roll plane. Simultaneously, the endolymph in the two sets of vertical semicircular ducts must eventually gain angular momentum, since they have been brought into a plane of constant rotation: until it does, however, the cupulae in the vertical ducts will be deviated, signaling that angular motion is occurring in the new planes of the vertical canals. The combined effect of the cupular deflections in all three sets of canals is that of a sudden angular velocity in a plane in which no actual motion is occurring. In the example given, the Coriolis illusion would be one of a sudden rolling to the left if the constant-velocity yaw were to the left and the pitching movement of the head were downward.

A particular phenomenon that occurs frequently in instrument flight in high-performance aircraft has been attributed to the Coriolis illusion

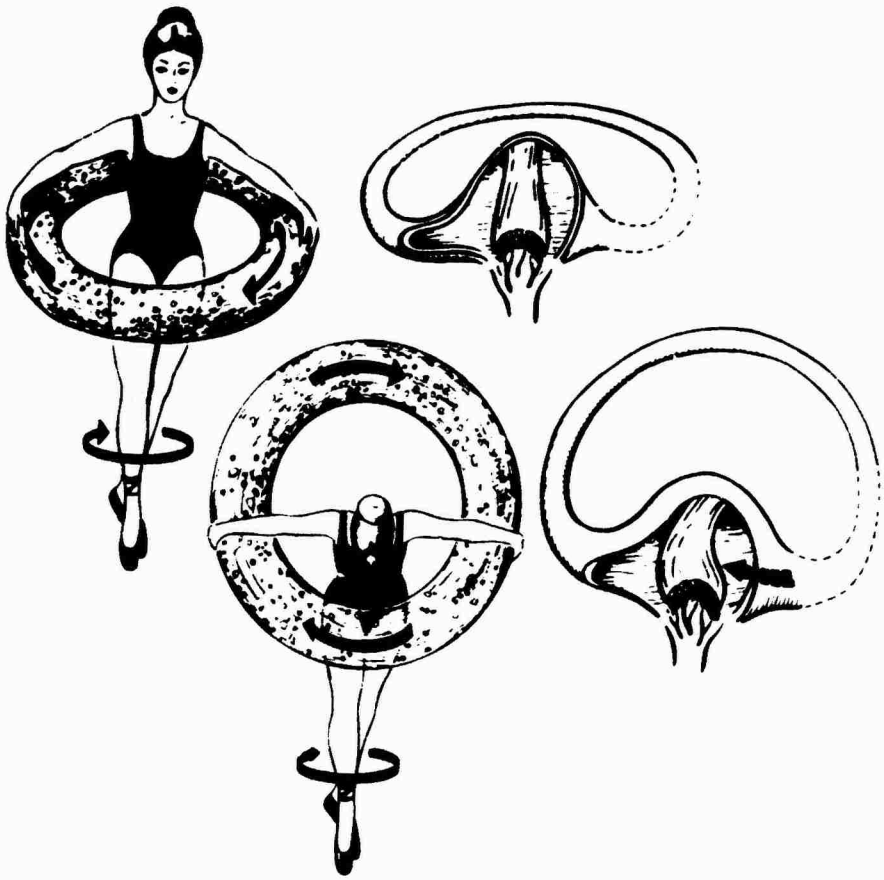


Figure 41. Mechanism of the vestibular Coriolis effect.

because it occurs in conjunction with large movements of the head during conditions of prolonged constant angular velocity. It consists of a very convincing sensation of rolling and/or pitching, which appears suddenly after the pilot has diverted his attention from the instruments in front of him and moved his head to view some knobs or switches in a relatively inaccessible part of the cockpit. This illusion is considered especially deadly, as it commonly occurs during the phase of flight prior to landing in which altitude is being lost rapidly and cockpit chores (e.g., radio frequency changes) are repeatedly requiring the pilot to break up his instrument cross-check. In a jet penetration turn, or any other routine in-flight maneuver, the existence of a great enough constant

angular velocity to cause a vestibular Coriolis effect has been questioned; and an alternative explanation for the false sensations generated by head movements in turning aircraft has been proposed. The fact that the high airspeeds of jet aircraft result in substantial $+G_z$ loads during moderate rates of turn suggests that the effect of moving one's head during such a turn is to rotate an abnormally long gravitoinertial force vector across the maculae; the result could be an abnormally large displacement of the otolithic membranes over their respective maculae, causing the false perception of an excessive angular displacement (tilt) of the head and body in space. The G-excess effect (also, abnormal-G effect), as the proposed phenomenon is called, is being examined at the time of this writing to determine whether it is responsible for what has been described in the past as the Coriolis illusion in flight.

Other vestibular illusions in flight are known definitely to be caused by stimulation of the otolith organs. Of these, the somatogravic (or posturogravic) illusion, which is a falsely perceived tilt of one's body with respect to the vertical, is the most commonly described; and it is all too frequently recorded as a cause of fatal aircraft accidents. (The term oculogravic illusion, used imprecisely in the past as a synonym for somatogravic illusion, refers to the illusory displacement of a viewed object which results when the otolith organs and somatosensory systems respond to rotation of the gravitoinertial force vector away from the vertical.) The somatogravic illusion occurs shortly after takeoff or during a missed approach, when full power has been applied to the aircraft and it is rapidly accelerating forward, causing a substantial backward inertial force to act on the pilot. When the inertial force combines with the 1 G of gravitational force acting on the pilot, the resultant gravitoinertial force is not only greater than 1 G, but what is even more important, the resultant force vector rotates backward to assume a new direction relative to that of the force of gravity. The otolithic membranes then shift their position on their maculae in response to the redirected inertial force, and the otolith organs signal the central nervous system that a condition similar to a backward tilt is occurring. As if to substantiate the false information coming from the vestibular system, the cutaneous and kinesthetic sensors also report the new direction of the gravitoinertial force. If the pilot allows himself to think, falsely, that "down" is in the direction of the resultant gravitoinertial force, his percept of the pitch attitude of his aircraft will change accordingly; and he may dive the aircraft in an attempt to level off from a normal pitch attitude which he has falsely perceived as being excessively nose-high (Figure 42). Full-power, shallow-angle crashes of high-performance aircraft, occurring

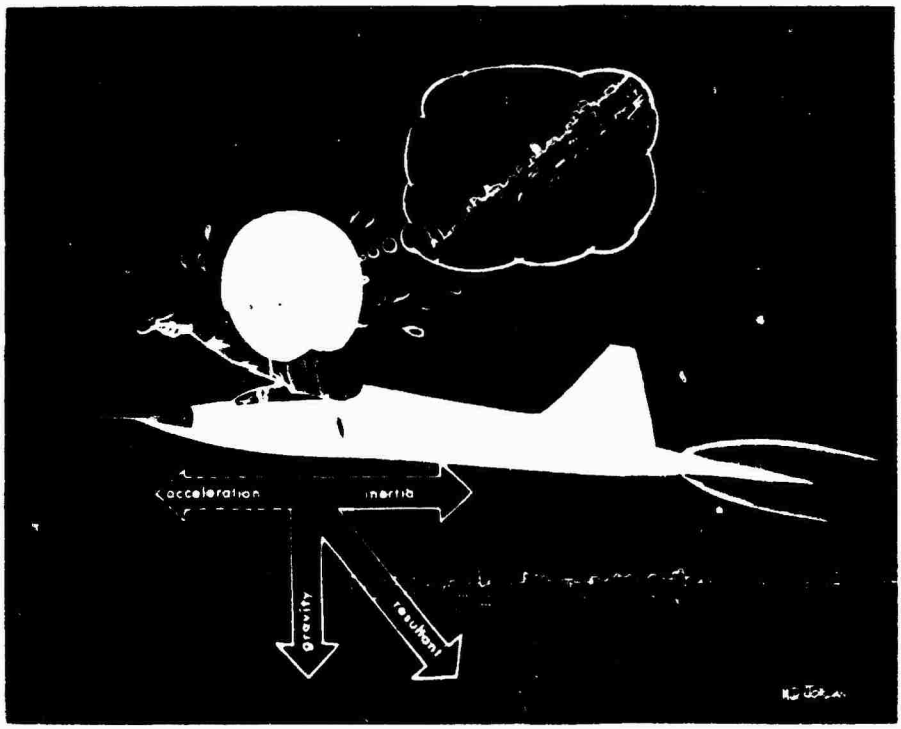


Figure 42. The somatogravic illusion as it occurs during forward linear acceleration.

within a few miles from the departure end of the runway in conditions of poor visibility, must always be suspected of having resulted from the somatogravic illusion.

The inversion illusion is akin to the somatogravic illusion, and some classify it as a subtype of same. It reportedly has occurred during level-off from a steep climb in low-visibility, turbulent conditions: apparently the $-G_Z$ centrifugal force resulting from the level-off and the $+G_X$ inertial force resulting from the tangential acceleration as the aircraft picks up speed combine with the gravitational force to form a resultant force vector which rotates backward and upward during the level-off (Figure 43). The resulting sensation of a backward tilt to the inverted position is reflexly countered by the pilot with a forward movement of the stick; this maneuver, however, merely intensifies the illusion, because it generates more centripetal and tangential acceleration as the aircraft pitches downward. The pilot, by continuing to respond inappropriately to this situation, can ultimately fly his aircraft into an aerodynamic condition (negative angle of attack) from which recovery is exceedingly difficult.

Figure 43. The inversion illusion--another form of the somatogravic illusion.

A final classic vestibular illusion resulting from otolith-organ stimulation is the elevator illusion, which results from the elevator reflex discussed earlier. If a pilot is subjected to an upward linear acceleration and experiences a $+G_z$ inertial force, as he might in turbulence or in a level-off after a steep descent, his eyes reflexly look downward and the instrument panel in front of him necessarily appears to move in the opposite, upward, direction. Thinking that the panel and the attached aircraft have pitched up, the pilot may correct for the illusory change of attitude and enter an unperceived dive.

A preponderance of emphasis has been placed on the vestibular mechanisms of illusions generated in flight, to the relative exclusion of the other parts of the proprioceptive system--the muscle, tendon, and joint receptors--and to the exclusion of the cutaneous receptors. These other sensors, however, are usually incapable of correcting misinformation that the vestibular system transmits, because they are

stimulated by the same mechanical energies (reactive forces and torques) that stimulate the vestibular system, and they generate perceptual errors very similar to those created by the vestibular system. The visual system, on the other hand, conveys information gathered from another form of energy (light); and to the extent that the visual environment provides an accurate representation of the true motional environment, spatial orientation can be maintained visually. This is done either by means of direct information, e.g., via a glimpse of the horizon, or by means of coded information gleaned from the flight instruments. Occasionally, however, the visual system is responsible for producing vertigo.

A common visual phenomenon associated with flying at night and in certain other conditions is misplacement of the horizon. This can happen when a pilot sees ground lights dotting a dark countryside and he confuses them with stars; or when he takes off over water on an overcast night in which the water blends with the sky, and the shoreline receding below the aircraft suddenly appears to be the horizon (Figure 44). Some pilots have even reported feeling inverted under such conditions; the very hazardous nature of this type of visual illusion should be obvious. When flying above the weather, pilots often feel compelled to align the wings of their aircraft parallel to the upper surface of the undercast, which may or may not be truly horizontal (Figure 45); this can result in a visual form of the leans, with a consequent annoying, if not dangerous, loss of flying efficiency. A similar condition often occurs during flight over sloping terrain, when the pilot fails to maintain straight and level flight because he misinterprets the sloped terrain as being horizontal.

Visual autokinesis is the illusory movement a small, stationary light exhibits when one stares at it for several seconds in the dark (Figure 46). As might be imagined, this phenomenon can be disturbing to a pilot trying to hold a steady position relative to another aircraft with a single position light. The autokinetic motion of a single star or planet reportedly can deceive a pilot into thinking he is closing on another aircraft, at least until he realizes he is making scant progress toward a join-up. The physiologic mechanism responsible for visual autokinesis is unknown. It has been found, however, that visual autokinesis becomes less pronounced with expansion of the visual framework: the bigger the light, the brighter the light, and the more lights that are visible to the observer, the less the autokinetic effect.

One powerful visual effect that can result in spatial disorientation is generated by shapes or patterns caught moving in the visual field,

Figure 44. Misplacement of the horizon--blending of earth and sky.

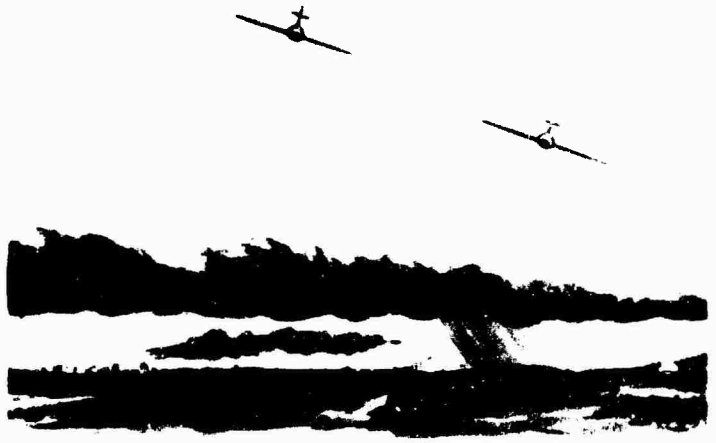


Figure 45. Misplacement of the horizon--sloping-cloud-deck phenomenon.

Figure 46. Autokinesis.

especially the peripheral field. The effect is a visually induced sense of self-motion: if a false sense of rotation is generated, it is called circularvection; and if a feeling of linear movement results, it is termed linearvection. An example of circularvection is the sense of turning and concomitant disorientation that can result when a pilot sees the revolving reflection of the beam from his rotating anticollision light as he flies through the clouds at night. Nearly everyone who drives an automobile has experienced linearvection: when stopped for a stoplight and the adjacent car creeps forward, one may suddenly slam on the brakes to arrest the falsely perceived backward motion of his own car. The undulating northern lights possibly cause visually induced sensations of self-motion that contribute to the vertigo commonly reported during night-time aerial refueling in northern latitudes; similar sensations are possibly elicited by descending target-illuminating flares that purportedly cause disorientation during night-time close air support missions. False cues of verticality can also be generated by northern lights and flares, however, and such false cues might underlie an alternative mechanism for the reported disorientation.

Other perceptual and behavioral phenomena occasionally occur in flight and are sometimes discussed in conjunction with visual causes of spatial disorientation. Fascination, target hypnosis, and the breakoff phenomenon are actually altered states of consciousness, not spatial disorientation, and will not be discussed in this review. Nor is flicker vertigo truly vertigo in the aeromedical sense, but an altered state of consciousness caused by a rapidly flashing light, such as sunlight viewed through a helicopter rotor or an idling propeller.

The interesting thing about spatial disorientation is that a particular set of linear or angular accelerations or misleading visual cues will not always produce illusory phenomena. When adequate external visual reference is available, spatial disorientation simply does not happen, despite the presence of linear or angular accelerations that can produce spatial disorientation when outside visual reference is excluded. This is a manifestation of visual dominance. In addition, even if outside visual reference is excluded, and the same erroneous vestibular cues are presented to a pilot on several different occasions, he will not necessarily experience an illusion on each occasion. When the pilot is extremely busy with cockpit chores, when he is anxious, when he is fatigued, when he is less proficient at instrument or formation flying than he should be, when he is mentally stressed for any reason, he is then more likely to become spatially disoriented in a given motional environment.

Normally, the instrument cross-check is little trouble to accomplish for an experienced pilot, whose visual dominance permits him to process the visual information from the instrument panel as true orientation information, to the exclusion of other, potentially disorienting, sensory data. In flight, the pilot is able to filter the information from his vestibular and kinesthetic sensors to provide himself with only the high-frequency, relatively accurate, motion-onset cues that make actual flying easier than controlling a static instrument procedures trainer. But when the pilot must repeatedly switch back and forth between an external visual reference and the instruments, or when the pilot is very busy with distracting cockpit duties, or when he is stressed by any of a multitude of things, his instrument cross-check becomes compromised or neglected. It is believed that the pilot, at these critical times, suddenly places his reliance upon all the natural, continuous, vestibular information that is available, rather than upon the coded, intermittently sampled, panel information. If this is allowed to happen,

sensory conflict--between the visual and vestibular systems--appears, and the pilot must somehow decide which sensory information to use in controlling the aircraft. This decision is not always easy, because acting on vestibular information is almost reflex, while acting on information presented by the instrument panel in the face of conflicting vestibular cues requires a very deliberate, concentrated effort. There is evidence that the central nervous system will not tolerate such a conflict for any appreciable length of time, and that resolution of the conflict takes place quickly in favor of the vestibular cues, without the benefit of the pilot's logical evaluation of the situation. In that case the sensory conflict may not be recognized as such: pilots have been known to transmit radio messages during presumed episodes of vertigo, claiming, "the aircraft control is goofed up," or "the attitude indicator isn't working." The giant hand phenomenon is known to occur in such situations: it is characterized by the pilot's feeling that someone or something is actively preventing his execution of the required corrective maneuver, either by pushing against the control stick when the maneuver is initiated or by countering every accomplished correction with a shove back to the original undesirable attitude. The giant hand is, in reality, the pilot acting against himself in a conflict raging between higher and lower levels of his central nervous system. In the extreme case, the pilot is completely oblivious to the fact that he is disoriented and flies into the ground without even realizing his error.

The fact that one's ability to orient by instruments is stress-sensitive leads to speculation about what can happen to a pilot once he has become disoriented. If the disorientation is itself a stress-provoking condition, the pilot might then give even more weight to his vestibular information, so that even more erroneous sensory cues would be received and incorporated into his false percept of spatial orientation: this would in turn create even more mental stress and anxiety. A condition of positive feedback would then exist, and it would theoretically be extremely difficult for the pilot to extricate the true percept of orientation and the equanimity with which he should operate. Under such a condition, a true panic state could conceivably result.

Preventive Measures

Given that man was designed to walk, run, and climb trees, but not to fly, what can be done to minimize the number of aircraft accidents caused by spatial disorientation? Indoctrination of pilots is the first

important step to take in the fight against spatial disorientation accidents. Lectures, demonstrations, and movies discussing sensory functions and the conditions under which they become inadequate should be provided for pilots by Physiological Training Officers and Flight Surgeons. Updating and improvement of training aids should be accomplished periodically to insure the adequate dissemination of pertinent knowledge to the pilot population. After all, "Forewarned is forearmed."

Some changes in the art and science of flying have resulted directly from analyses of spatial-disorientation accidents. For example, we are now aware of the problem caused by placing radio frequency selector knobs in positions where the pilot has to turn his head to change frequencies, and designers of modern aircraft try to place such knobs so that no extreme head movements are required for their operation. Attitude indicators have undergone substantial improvement with regard to their verisimilitude. The HUD (heads-up display) also appears to be a welcome improvement, in that it allows the pilot to remain visually oriented by aircraft instruments while simultaneously performing out-of-cockpit visual tasks. Several potentially dangerous flying practices (for example, instrument takeoffs and night formation rejoins) have been officially discouraged in most commands. Most certainly, however, additional safety measures must be conceived, proposed, deliberated upon, and put into effect, in the continuing effort to reduce spatial-disorientation accidents.

MOTION SICKNESS

Motion sickness is characterized by a progression of symptoms that occurs in conjunction with and in response to unaccustomed conditions existing in one's motional environment. Those symptoms are apathy, headache, stomach awareness, pallor, perspiration, nausea, vomiting, and prostration, usually in that sequence, and usually, but by no means always, concomitant with actual motion. Included in the generic term "motion sickness" are seasickness, airsickness, carsickness, trainsickness, motion-picture sickness, flight-simulator sickness, and a number of other species of this mysterious disease.

Importance of Motion Sickness

The major undesirable impact of motion sickness is in military endeavors. The following quotation from Armstrong provides some

interesting statistics associated with the World War II military effort:

> "It was learned that 10 to 11% of all flying students became airsick during their first 10 flights, and that 1 to 2% of them were eliminated from flying training for that reason. Other aircrew members in training had even greater difficulty and the airsickness rate among them ran as high as 50% in some cases. It was also found that fully trained combat crews, other than pilots, sometimes [developed airsickness] which affected their combat efficiency. An even more serious situation was found to exist among airborne troops. Under very unfavorable conditions, as high as 70% of these individuals became airsick and upon landing were more or less temporarily disabled at a time when their services were most urgently needed."

Motion sickness also took its toll among amphibious assault troops in World War II: a very high percentage of these troops were motion sick upon arrival at the beach landing area unless they were allowed to look over the edge of the vehicle during its journey to the beach.

More recent studies of the incidence of airsickness in flight training reveal that approximately 40% of all aircrew trainees become airsick at some time during their training, and that approximately 18% of student pilots develop motion-sickness symptoms severe enough to interfere with control of the aircraft. These symptoms appear with greatest frequency during the first several training flights, the first spin-training flight, and during the first few dual aerobatic flights, which should come as no surprise to the reader. The adaptation of which most people are capable is evidenced in the fact that only about 1.4% of USAF student pilots and about 0.7% of student naval aviators have to be eliminated from flying training because of intractable airsickness. Although recent data on student navigators are not available, the figures from World War II suggest that perhaps 65% of these individuals develop airsickness, and as high as 5% must be eliminated from training because of it.

Although test pilots and astronauts, probably as a result of selection, are in general quite resistant to motion sickness, our experience to date with space flight reveals there is a definite risk that astronauts will become motion sick in the weightless environment. Several Skylab astronauts reported symptoms of motion sickness for a few days after beginning

their long missions at zero G; we also know that a number of cosmonauts became motion sick in the early days of the Soviet space program. Even though the cardiovascular decompensation associated with a prolonged stay in a weightless environment would theoretically be minimized in a satellite that is rotated to provide artificial gravity, a substantial problem with motion sickness must be anticipated in the event such a system is used, because extremely nauseating Coriolis effects are incumbent in such rotating environments.

For decades it has been popular to try to determine as fully as possible the particular qualities of motion that are responsible for motion sickness, and even to try to isolate a particular set of sensory end-organs responsible. As a result, we have much useful information about how the vestibular system is stimulated and how it responds, but the pathogenesis of motion sickness has eluded definition. To be sure, abnormal stimulation of the semicircular-duct system, as by a rotating chair or rotating room, can cause motion sickness; Coriolis effects are especially potent in this regard. Likewise, abnormal stimulation of the otolith organs, as by a rapidly accelerating elevator or a heaving ship deck can result in motion sickness. In aircraft in aerobatics, human centrifuges, life rafts or space capsules bobbing on the ocean, and many other vehicles capable of complex motions, both the semicircular-duct system and the otolith-organ system are abnormally stimulated, and motion sickness is the result. Of particular interest is the fact that the absence of functioning vestibular end-organs confers a complete immunity to motion sickness upon any individual unfortunate enough to be so deprived, which indicates that the vestibular system is somehow of primary importance in the pathogenesis of motion sickness.

Movement sensed solely through the visual system can also lead to motion sickness. Motion pictures of roller-coaster rides and ships in rough seas nauseate some people, and certain highly susceptible individuals can even make themselves motion sick while scanning slides under a microscope. A substantial incidence of motion sickness was reported in the Navy 2-FH-2 helicopter hover trainer, a static device which portrayed motion by means of a projected visual reference: the most interesting fact was that there was a difference in susceptibility between instructors and students, the instructors exhibiting a much greater incidence of motion sickness in the device. One's visual reference does not necessarily even have to move for him to develop motion sickness: "anti-gravity" houses in amusement parks, constructed on a slant

so that the apparent vertical and the gravitational vertical are noticeably different, can cause motion sickness in particularly susceptible individuals. The role of the visual system in the etiology of motion sickness is further exemplified by the well-known fact that absence of an outside visual reference makes persons undergoing abnormal motion more likely to become sick than they would be if an outside visual reference were available. Cases in point are the pilot who gets sick while riding as a passenger in turbulent conditions, and the sailor who comes topside to view the horizon in an attempt to prevent the progression of motion sickness symptoms.

In fact, it appears that the abnormal stimulation of any sensory system which provides spatial orientation information, be it vestibular, visual, kinesthetic, auditory, or tactile, can result in motion sickness, providing the vestibulocerebellar axis is intact. Furthermore, fear and insecurity, whether about one's general well-being or about one's orientation in space, are also definitely related to motion-sickness susceptibility. One study even indicates that the personality characteristics of emotional lability and excessive rigidity are positively correlated with motion-sickness susceptibility. Such things as mechanical irritation of the viscera and malodorous aircraft compartments, however, although they are commonly associated with conditions that result in motion sickness, do not in themselves cause motion sickness.

How can the volumes of scientific data and numerous anecdotes dealing with motion sickness be incorporated into a coherent theory? At present they cannot, at least not to the complete satisfaction of all who study in this field. There is general agreement, however, that incongruous sets of sensory data related to spatial orientation are necessary for the production of motion sickness, and at least one of those sets of data must originate in the vestibular organs. It is also agreed that the cerebellum plays a critical part in the production of motion sickness, but how it does so is disputed. The potentiating effect of emotional arousal and heightened vigilance is also accepted, but incorporated into different theories in different ways.

One of the older theories of the pathogenesis of motion sickness was the so-called "cerebellar-sweating" theory. It proposed that vestibular stimulation causes certain vestibular projection areas of the cerebellar cortex (nodulus, uvula) to secrete a neurohumor which traverses the fourth ventricle and stimulates the underlying medullary

chemoceptive emetic trigger zone. A more recent theory suggests that sensory conflict relating to spatial orientation causes the cerebellum to initiate a demand for more information about the acceleratory environment from the vestibular sensors, which information is provided by a relaxation of the normal amount of vestibular suppression exerted by the vestibular efferent system; the augmented flow of vestibular signals then "spills over" from the vestibular nuclei into the contiguous motor nuclei of the vagus nerve, resulting in symptoms of motion sickness. A third theory is based on the assumption that vascular reflexes of vestibulocerebellar origin normally occur in response to anticipated motion: Steele states that, in motion sickness, "the major symptoms seem to be caused by cardiovascular inadequacy, secondary to diversion of circulating blood to the muscles in response to a threatened need for vigorous muscular action on the basis of inadequately perceived inertial and dynamic environment." Other theories have been proposed, but usually their emphasis on vestibular mechanics has rendered them too narrow to support the data relating to visually induced motion sickness and the potentiating effect of apprehension. This writer finds an amalgamation of the second and third theories mentioned above to provide a satisfactory explanation of the cause of motion sickness, at least for the present.

Prevention and Treatment

Once the symptoms of motion sickness have developed, treatment generally tends to be ineffective. The first step to be taken, obviously, is to stop the motion of the vehicle; if this is impossible, the patient should be advised to lie down, or at least to keep his head still, while the motion continues. Flight instructors can sometimes avert airsickness in student pilots by discontinuing in-flight demonstrations and turning over control of the aircraft to the student. Sometimes providing the patient with a stable external visual reference, like the earth's horizon, will prevent the progression of symptoms: the sailor who comes topside to thwart impending seasickness exemplifies this form of treatment. Another procedure which has proved useful in practice is to cool the patient with a blast of air from the air conditioner or cabin air vent; such thoughtfulness on the part of their instructors has undoubtedly saved many aviation cadets the embarrassment of having to clean up the cockpit.

After symptoms have appeared, it is useless to try to stop their progression by giving oral medications. Injections of diazepam, 5 to

10 mg I.V., or scopolamine, 0.5 mg I.V., have been effective in arresting motion sickness in some cases. Scopolamine nasal spray or nose drops, and various antihistaminic suppositories have also been used with some degree of success. In other circumstances it may be necessary to give intravenous phenobarbital or even morphine to prevent a progressive deterioration of the patient's condition.

Much more rewarding than after-the-fact treatment of motion sickness is prophylaxis. The most effective medical countermeasure against motion sickness is scopolamine, 0.3 to 0.6 mg, combined with dextroamphetamine, 5 mg, taken orally one hour prior to exposure to motion. Promethazine, 25 mg, and dextroamphetamine, 5 mg, is almost as effective a combination, and may be preferable because of the lesser severity of side effects. Repeated doses of either combination may be necessary in prolonged exposures to offending motion, such as a voyage at sea. Meclizine, 50 mg, or cyclizine, 50 mg, taken orally one hour before exposure, are the traditional medications used to prevent motion sickness, but their effectiveness is quite low when compared with that of the "scope-dex" combination mentioned above.

The use of anti-motion-sickness medications in pilots and other aircrew requires a judgment by the Flight Surgeon on whether the benefits of the medication will outweigh the risks resulting from side effects of the drugs. As a rule, the pilot with primary responsibility for control of an aircraft should never take anti-motion-sickness medications. This does not, however, preclude giving student pilots prophylactic medications for particular dual training flights in which airsickness is likely to degrade performance and learning efficiency. If it appears that a student pilot will have a problem with airsickness, it is advisable to provide him with a modicum of pharmacologic protection while he habituates to the motion-sickness-producing stimuli; this will prevent the development of <u>conditioned motion sickness</u>, an unfortunate state characterized by symptoms of motion sickness evoked by the mere sight of a vehicle which repeatedly caused actual motion sickness on past occasions (Figure 47).

Although various screening techniques (questionnaires and tests of vestibular function) have been developed which purport to indicate the probability that a certain person will develop airsickness during training, there is presently no justification for preventing one from undertaking training on the basis of results of such screening. The reason for this is that most people adapt or habituate to motion-sickness-producing stimuli to the point where they can effectively carry out required

military tasks. If it becomes apparent that the trainee is not adapting satisfactorily to the abnormal acceleratory environment, then the Flight Surgeon should evaluate him, assess the emotional and motivational factors that may be involved in the case, and seek psychiatric consultation if necessary. Those chronically airsick students who are found to have an unquestionable motivation to fly should be considered for the Motion Sickness Rehabilitation Program at the School of Aerospace Medicine. The two-week program involves twice-a-day sessions of self-paced Coriolis stimulation to the point of pallor and perspiration, combined with daily one-hour behavior modification exercises involving systematic desensitization (to counter the anticipatory anxiety that attends repeated episodes of motion sickness). Calisthenics and a great deal of positive reinforcement are also included. Since the results of the program to date indicate a success rate in excess of 85%, it may be practical to attempt to reclaim some students who would otherwise surely wash out.

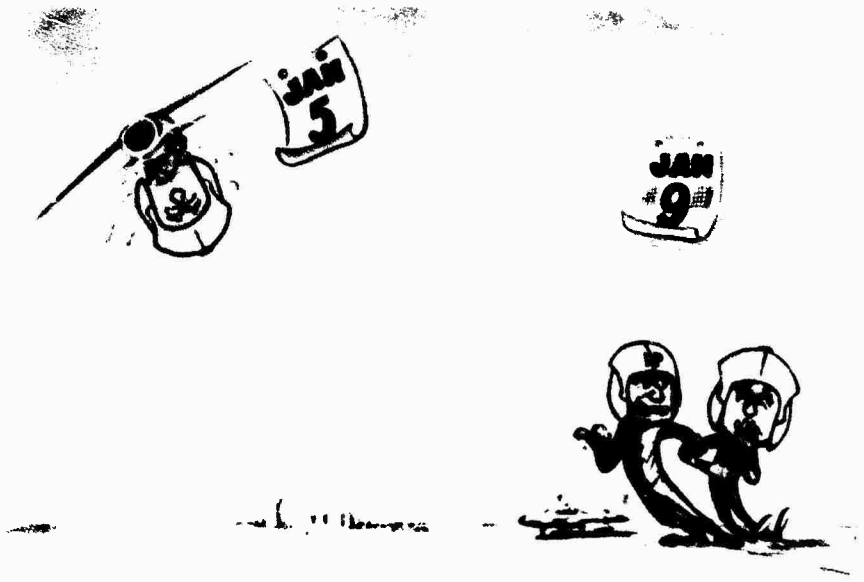


Figure 47. Conditioned motion sickness.

BIBLIOGRAPHY

Linear and Angular Motion

Cannon, R. Dynamics of physical systems. New York: McGraw-Hill Book Company, 1967.

Dixon, F., and J. Patterson. Determination of accelerative forces acting on man in flight and in the human centrifuge. U.S. Naval School of Aerospace Medicine monograph, 1953.

Fryer, D., and E. Evrard (eds.). Glossary of aerospace medical terms. AGARDograph 153, Advisory Group for Aerospace Research and Development, North Atlantic Treaty Organization, 1971.

Gell, C. Table of equivalents for acceleration terminology, recommended for general international use by the Acceleration Committee of the Aerospace Medical Panel, AGARD. Aerosp Med 32:1109 (1961).

Hixson, W., J. Niven, and M. Correia. Kinematics nomenclature for physiological accelerations, with special reference to vestibular applications. Monograph 14, Naval Aerospace Medical Institute, 1966.

Kaufman, W. C. Compilation of information for authors of Aerospace Medical manuscripts. Aerosp Med 44:1213 (1973).

Short-Duration $+G_z$, High Sustained $+G_z$, Effects of $-G_z$, Effects of $\pm G_x$

Burns, J. Reevaluation of a tilt-back seat as a means of increasing acceleration tolerance. Aviation, Space and Environmental Med 46:55 (1975).

Burton, R., and J. Jaggars. Influence of ethyl alcohol ingestion on a target task during sustained $+G_z$ centrifugation. Aerosp Med 45: 290 (1974).

Burton, R., and R. Krutz. G tolerance and protection with anti-G suit concepts. Aviation, Space and Environmental Med (In press).

Burton, R., S. Leverett, and E. Michaelson. Man at high sustained $+G_z$ acceleration. AGARDograph 190, Advisory Group for Aerospace Research and Development, North Atlantic Treaty Organization, 1974.

Burton, R., M. Parkhurst, and S. Leverett. $+G_z$ protection afforded by standard and preacceleration inflations of the bladder and capstan type G-suits. Aerosp Med 44:488 (1973).

Cochran, L., P. Gard, and M. Norsworthy. Variations in human G tolerance to positive acceleration. Research Report NM001 059.02-10, U.S. Naval School of Aviation Medicine, 1954.

Duane, T. Observations on the fundus oculi during blackout. Arch Ophthalmol 51:343 (1954).

Fraser, T. Sustained linear acceleration. In Bioastronautics Data Book, Special Publication 3006, ch. 4. National Aeronautics and Space Administration, 1973.

Gauer, O. The physiological effects of prolonged acceleration. German Aviation Medicine, World War II, vol.I. Washington, D.C.: U.S. Government Printing Office, 1950.

Gauer, O., and G. Zuidema (eds.). Gravitational stress in aerospace medicine. Boston: Little, Brown and Co., 1961.

Henry, J., et al. Factors maintaining cerebral circulation during gravitational stress. J Clin Invest 30:292 (1951).

Henry, J., and J. Meehan. The circulation: an integrative physiologic study. Chicago: Year Book Medical Publishers, Inc., 1971.

Howard, P. Accelerations. In J. Gillies (ed.). A textbook of aviation physiology. Oxford: Pergamon Press, 1965.

Krutz, R., S. Rositano, and R. Mancini. A comparison of techniques for measuring $+G_z$ tolerance in man. J Appl Physiol (In press).

Leverett, S., et al. Physiologic responses to high sustained $+G_z$ acceleration. SAM-TR-73-21, Dec 1973.

Leverett, S., and W. Newsom. Photographic observations of the human fundus oculi during $+G_z$ blackout on the USAF School of Aerospace

Medicine centrifuge. In J Lunc (ed.). 19th International Astronautical Congress of Bio-Astronautics, Book 4. Oxford: Pergamon Press, 1971.

Lindberg, E., and E. Wood. Acceleration. In J. Brown (ed.). Physiology of man in space. New York: Academic Press, 1963.

Parkhurst, M., S. Leverett, and S. Shubrooks. Human tolerance to high, sustained $+G_z$ acceleration. Aerosp Med 43:708 (1972).

Rogers, D., et al. Effect of modified seat angle on air to air weapon system performance under high acceleration. USAF-AMRL-TR-13-5, 1973.

Shubrooks, S., and S. Leverett. Effect of the Valsalva maneuver on tolerance to $+G_z$ acceleration. J Applied Physiol 34:460 (1973).

Stoll, A. Human tolerance to positive G as determined by the physiological end points. J Aviat Med [Now Aerosp Med] 27:356 (1956).

Perception of Motion and Position, Spatial Disorientation, and Motion Sickness

Armstrong, H. Airsickness, ch. 14. In H. Armstrong (ed.). Aerospace medicine. Baltimore: Williams & Wilkins, 1961.

Barnum, F. Spatial disorientation aircraft accidents, 1 Jan 1969-31 Dec 1971. Presented at the 44th Annual Scientific Meeting of the Aerospace Medical Association, Las Vegas, Nev., May 1973.

Barnum, F., and R. Bonner. Epidemiology of USAF spatial disorientation aircraft accidents, 1 Jan 1958-31 Dec 1969. Aerosp Med 42:896 (1971).

Benson, A. Spatial disorientation in flight. In J. Gillies (ed.). A textbook of aviation physiology, ch. 40. Oxford: Pergamon Press, 1965.

Benson, A. (ed.). The disorientation incident. AGARD Conference Proceedings 95 Part I, Advisory Group for Aerospace Research and Development, North Atlantic Treaty Organization, 1972.

Benson, A., and E. Burchard. Spatial disorientation in flight--A handbook for aircrew. AGARDograph 170, Advisory Group for Aerospace Research and Development, North Atlantic Treaty Organization, 1973.

Chinn, H., and P. Smith. Motion sickness. Pharmacol Rev 7:33 (1955).

Dobie, T. Airsickness in aircrew. AGARDograph 177, Advisory Group for Aerospace Research and Development, North Atlantic Treaty Organization, 1974.

Gillingham, K. A primer of vestibular function, spatial disorientation, and motion sickness. SAM Aeromed Rev 4-66, June 1966.

Howard, I., and W. Templeton. Human spatial orientation. New York: John Wiley and Sons, 1966.

Jones, G. Vestibulo-ocular disorganization in the aerodynamic spin. Aerosp Med 36:976 (1965).

Kellogg, R. Appendix A: USAF spatial disorientation accidents during the period 1968-1972. In Staff study report on spatial orientation training. Aerospace Medical Division, Brooks AFB, Texas, Dec 1974.

Money, K. Motion sickness. Physiol Rev 50:1 (1970).

Moser, R. Spatial disorientation as a factor in accidents in an operational command. Aerosp Med 40:174 (1969).

Nuttall, J. The problem of spatial disorientation. JAMA 166:431 (1958).

Nuttall, J. B., and W. G. Sanford. Spatial disorientation in operational flying. Publication M-27-56, U.S. Air Force Directorate of Flight Safety Research, Sept 1956.

Peters, R. Dynamics of the vestibular system and their relation to motion perception, spatial disorientation, and illusions. Contractor Report 1309, National Aeronautics and Space Administration, 1969.

Steele, J. The symptomatology of motion sickness. In Fourth Symposium on the Role of the Vestibular Organs in Space Exploration. Special Publication 187, National Aeronautics and Space Administration, 1970.

Tyler, D., and P. Bard. Motion sickness. Physiol Rev 29:311 (1949).

Wood, C., A. Graybiel, and R. Kennedy. Comparison of effectiveness of some anti-motion-sickness drugs using recommended doses as tested in the Slow Rotation Room. Aerosp Med 37:259 (1966).

Young, L., et al. Interaction of optokinetic and vestibular stimuli in motion perception. Acta Otolaryngol (Stockh) 76:24 (1973).